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A TREATISE

ON THE

PRACTICE OF MEDICINE

FOR THE USE OF

STUDENTS AND PRACTITIONERS OF MEDICINE

BY

ROBERTS BARTHOLOW, M. A., M. D., LL. D.,

Professor of Materia Medica, General Therapeutics, and Hygiene, in the Jefferson Medical College of Philadelphia; formerly Professor of the Theory and Practice of Medicine in the Medical College of Ohio, Cincinnati; Fellow of the College of Physicians of Philadelphia; Member of the American Philosophical Society; Fellow of the Royal Medical Society of Edinburgh, of the *Société Médico-Pratiques de Paris*, of various Medical Societies, American and State; Prize Essayist of the American Medical Association for 1868, of the Connecticut (Russell and Jewett), and of the Rhode Island State Medical Societies; Author of a Practical Treatise on Materia Medica and Therapeutics, of a Treatise on Medical Electricity, a Manual of Hypodermatic Medication, etc.

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SEVENTH EDITION, REVISED AND ENLARGED

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1890

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I DEDICATE THIS TREATISE

TO THE MEMORY OF MY DEAR FRIEND

R. R. SPRINGER,

LATE OF CINCINNATI, OHIO.

PREFACE TO THE SEVENTH EDITION.

In sending forth another edition of this work, it would be a mere affectation if I failed to give utterance to my sense of the remarkable appreciation shown for it by medical readers. No higher compliment can be paid an author than the purchase, distribution, and reading of successive editions of a bulky volume, nor can an author make a better return for such a high estimate of his work than a sincere effort to so prepare each issue as to make it more worthy of regard than were its predecessors. Accordingly, I have incorporated all the improvements in medical science and art that have stood the tests of prolonged and painstaking observation and experience. To do this the number of pages has been somewhat increased, notwithstanding in the new material as in the old the utmost conciseness, consistent with clearness and accuracy, has been the guiding principle in the composition of the text.

Roberts Bartholow, M. D.

Philadelphia, 1527 Locust Street.

PREFACE TO THE SIXTH EDITION.

In preparing this edition of my Treatise on the Practice of Medicine, I have sought to make it still more worthy the approbation of my readers. In no way can this purpose be more certainly assured than by increasing its practical resources. Not overlooking the advances made in scientific medicine, I have, however, devoted most attention to the clinical aspects of the subject. In developing this purpose, I have tried to preserve that harmony and proportion, in the treatment of the various topics, so necessary in a systematic treatise. It has been imperative, to keep in view my original design, to prepare a work in three volumes, which should cover the whole domain of special pathology and therapeutics.

I had not dared to mention so ambitious a purpose until two of these volumes—Materia Medica and Therapeutics, Practice of Medicine—had appeared and been favorably received. The third of this series, intended to treat of the Principles of Medicine, is now in course of that careful and deliberate preparation necessary in the treatment of such an important subject. While each one is an independent work, they are expected to be mutually complementary. In executing so large an undertaking, it is necessarily difficult to preserve a proper proportion in its various parts. I can not flatter myself that I have entirely overcome this difficulty.

In the present edition some new subjects have been introduced, and preliminary chapters have been appended to the chief divisions of the work, to make the study of the diseases of the class more exact, and to enhance the practical character of the whole. I believe I may say that little, properly pertaining to the domain of practice, has been overlooked, and that nothing superfluous has been admitted. That the work might have been written from a different standpoint is quite true, but then its character had been changed. As the purpose was to prepare a concise, practical treatise, to be independent in itself, and yet form a part of a larger whole, the subject must necessarily be discussed as has been done here. That some of my critics think differently, because they have conceived another ideal, is inevitable, but any regret I might have over this disapproval of my plans is quite tempered by the reflection that I am not striving to placate such critics so much as to please myself, and especially the large body of my sympathetic readers, whose approval is testified in the disappearance of successive large editions of the work. To them I repeat the assurance of my gratitude for the favor with which they have received my contributions to the Practice of Medicine.

R. B.

PHILADELPHIA, 1527 LOCUST STREET,
July 1, 1886.

PREFACE TO THE FIFTH EDITION.

Again there is imposed on me the grateful duty of acknowledging my indebtedness to numerous readers for their unfailing appreciation of my work.

The fifth edition has followed so soon on the fourth that but few changes are necessary; yet these happen to be important. The bacillus tuberculosis has been discovered, and minute organisms are increasing in pathogenetic importance. Any work presuming to represent the present state of medical knowledge can not be unmindful of such topics. Some other changes and additions have been made—in all, increasing the size of the work by about twenty pages.

In submitting this edition to the medical public, I can do no less than reiterate the expression of my hope that it may continue to enjoy the favor bestowed on the work from its first issue.

Roberts Bartholow.

1509 Walnut Street, Philadelphia,
April 15, 1883.

PREFACE TO THE THIRD EDITION.

If the rapid sale of large editions of a work may indicate that it supplies the needs of many readers, I ought to be satisfied with the measure of success to which this treatise has attained. That it is fulfilling its mission is demonstrated to me in the encouraging, even enthusiastic expressions of approval, coming from numerous correspondents, who have found in this work a safe guide and judicious counselor. It is especially gratifying that those who have followed modes of treatment here inculcated have not been disappointed in the results. This fact seems to be a sufficient answer to those pessimistic critics who have found the author over-sanguine and dogmatic in his statements on therapeutical points.

That the confidence and esteem of my readers may continue to be justified, I have sought, in this edition, to make my work more thorough by adding some new articles, and by subjecting the whole to a careful revision. About fifty pages have been added, thus enlarging the scope, and, I hope, increasing the utility of the book. It does not yet approximate in its dimensions to the standard of those critics who estimate a work according to its weight, or to the number of its pages. As the author despairs of propitiating the favor or of conforming to the views of such critics, he must needs bear as best he can the weight of their displeasure. He is, however, supported in the midst of this trial by the circumstances that his work has been very fa-

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The author has been furnished by various correspondents with the results of treatment, as obtained by them from the methods prescribed in the therapeutical sections. It is desirable to accumulate facts, and hence failures should be reported as well as successes. It is also desirable to ascertain if the results of drug administration correspond to the pretensions of therapeutists. Is the optimism of the positive school justified by the deductions of clinical experience? I hope that those of my readers who are making observations will publish them, in the interests of scientific truth, to silence the cavils of therapeutical nihilists on the one hand, or the claims of therapeutical optimists on the other.

ROBERTS BARTHOLOW.

1509 WALNUT STREET, PHILADELPHIA,
January, 1882.

PREFACE TO THE SECOND EDITION.

In less than one month after the publication of the first edition of three thousand copies of this treatise, the publishers called on me to prepare a second edition. This result is the more surprising, as it was accomplished before the numerous medical journals of the country had introduced the book to their readers, and pronounced a judgment on its merits or defects. I feel deeply grateful to my numerous readers for this substantial evidence of their appreciation of my labor. I have, also, a great many correspondents, in all parts of the country, to thank for kind expressions of approval, and for valuable suggestions.

In this edition I have corrected some typographical errors which escaped notice, and have amended some doubtful statements, and also have added articles on Alcoholism and on Sunstroke—topics overlooked in preparing the first edition. I have, further, enlarged the index, and have added an index of authors. In the first preface it was stated that I had avoided an attempt at bibliographical display, which has perplexed some of my bibliolatrous critics. One of them, in a notice of the work, was pleased to observe that I had condensed my materials from the text-books and cyclopædias. While expressly disclaiming an attempt to give a complete, even a full bibliography, I have indexed the authors referred to in the body of the work, which will show that I went to original sources for my information. Almost all of the works, monographs, and articles referred to, are contained in my own library of three thousand medical volumes; and those not in my own were obtained from the library of the College of Physicians of this city. I wish my book, however, to be regarded, as it should justly be, the product of my own study, observation, and experience. An author with any training in the methods of authorship will agree with me, that to prepare a work, every detail of which the writer has made his own, is greatly more laborious than merely compiling a work from abundant bibliographical resources. I could more easily have prepared two volumes from the materials at hand, than write this book; but it was not my purpose to merely compile a book on practice—rather to prepare one which contained my own conception of the subject. I venture to express the conviction that, the more carefully this work is examined, the more it will be found to contain the most recent and approved facts of special pathology and therapeutics, without verbose dilution, and literary and typographical padding. With the hope that the second edition will continue to enjoy the favor accorded to the first, I submit it to the discriminating judgment of the medical profession.

ROBERTS BARTHOLOW.

PHILADELPHIA, 1509 WALNUT STREET,
November 1, 1880.

PREFACE.

In undertaking the preparation of a treatise on the Practice of Medicine, I had the intention to write a companion volume to my work on Materia Medica and Therapeutics. When announced, the book was so far advanced that its completion was confidently anticipated within the year. Unfortunately, the condensation of material found necessary, when the work had reached that stage where its proportions could be judged with some accuracy, involved much additional labor. This was the more regrettable, as the incessant demands of a large private practice and the onerous duties of an exacting professorial position permitted little of that uninterrupted leisure which is essential for successful literary composition. Slow progress was inevitable under these circumstances, and hence it was not until my removal to Philadelphia last year that I could devote some hours each day to my arduous task. I trust that this explanation of the delay in the appearance of the treatise will be satisfactory to my readers, especially to the large number who have honored me by subscribing for the work long in advance of its publication.

As my treatise on Materia Medica and Therapeutics embraced those topics of importance to the physician, and omitted matters of rather extraneous interest, so in the preparation of this volume my purpose was to include the subjects embraced under the title of "Practice of Medicine," omitting those topics of general pathology, etiology, etc., with which the works on Practice usually open, and which, though sufficiently valuable in themselves, are too often passed over hastily, or not read at all, in the desire to reach the practical subjects. I have therefore omitted the topics in question from their position as an introduction to special pathology, and have, although at the disadvantage of some repetition, incorporated them in their proper relation with individual diseases.

That I should, under all the circumstances above stated, have undertaken such a task as the preparation of this treatise, for which, it may be suggested, there was no special need, and, if the need existed, there was no claim on me to supply it, may be accounted for by the fact that, when the work was begun, I was Professor of the Theory and Practice of Medicine and of Clinical Medicine in the Medical College of Ohio, and was urged, not only by the students and practitioners who attended my lectures, but also by many readers of my therapeutical treatise, to prepare a volume on Practice, which should have the practical characteristics, the definiteness of statement, the conciseness, and, at the same time, the fullness of the work on Materia Medica and Therapeutics. I was the more inclined to accede to these wishes because of a natural desire to appear as an author on subjects to which I had devoted all the years of my professional life, and under the most varied conditions. Serving as an officer of the medical staff of the United States Army in Kansas, Utah, Colorado, New Mexico, Minnesota, and during the war of the rebellion at Washington, Nashville, Chattanooga, Baltimore, etc., followed by an extensive practice (private and hospital) of sixteen years at Cincinnati, I may justly claim to have enjoyed large opportunities for the clinical study of the diseases of the North American Continent. With one or two unimportant exceptions, I have had personal charge of the maladies treated of in this work, and have made them the subject of clinical demonstration or *post-mortem* investigation, either privately or in public lectures.

In the treatment of the various topics, I have attempted to give to each just that amount of consideration to which its importance entitles it, within the limitations imposed by the size of the work. A just harmony and proportion can be secured only by condensing some subjects and displaying others. No space has been given to merely historical disquisitions, or to the discussion of controverted points. Also, to utilize all available space, chapters have been dispensed with, and the intervals between the sections have been abbreviated as much as possible. Notwithstanding my utmost efforts at condensation, the work has grown beyond the contemplated size; but I would fain hope that no part of it could be sacrificed without impairing the value of the whole.

Much of the matter embraced in a work of this kind is the common property of the medical profession, and hence I have not quoted many authorities. I have rather avoided references when their mention would have been mere pedantry, and would have occupied valuable space. Nevertheless, when I was distinctly indebted for some fact or opinion, I have given the reference to the authority. Sometimes, when the authority is well known, the name is merely inclosed in parentheses. It is a comparatively easy task, especially with the aids now at our disposal, to give an extended bibliography, but the space occupied would have swollen this work to encyclopedic proportions, without adding to its real utility. When an author only expresses the opinions of his authorities, he avoids the appearance of dogmatism, which must be the tone of a work giving utterance to individual opinions; but I could hardly do otherwise than draw my clinical material—the descriptions of diseases—from my own observations at the bedside. Also, a large experience in the treatment of disease could not fail to develop some positive convictions as to the real value of remedies. The reader will find that I have no sympathy with the therapeutical nihilism of the day, and that my convictions find expression in the recommendation of plans of treatment. In a work of this kind, intended for the guidance of young practitioners and students, some dogmatism, although offensive to the highest taste, may be pardoned, in view of the practical advantages of experienced leadership. Indeed, there is no department of the subject in which it seemed to me so necessary to express positive opinions. The influence of some of our most prominent medical thinkers has been opposed to the value of medicines in the treatment of disease. The modern school of pathologists, absorbed in the contemplation of the ravages of diseases, are either oblivious of the curative powers of remedies, or openly ridicule the pretensions of therapeutists. I have, therefore, in the therapeutical sections, especially endeavored to set forth true principles, and have taught the utility of drugs when rightly administered, but have none the less tried to indicate the limits of their utility, for he who is unmindful of the injury done by ill-directed or reckless medication is as unsafe a guide as the most pronounced therapeutical nihilist.

The pathological doctrines inculcated in the work are derived from the highest sources. The few illustrations of morbid changes introduced were obtained from the admirable atlas of Thierfelder. As my information on this subject was derived from those best qualified to instruct, I have not hesitated to express with some decision the present state of knowledge in respect to the pathology of the various diseases, desiring in this, as in other departments of my subject, to give some positive views. I may be criticised with the observation that, in the progress of discovery, the doctrines at present received unreservedly may be entirely overthrown, and very different views be substituted. It will be time enough, however, when the change comes, to adapt our opinions to the new order of pathological doctrines.

Having thus explained my intentions in producing the work, I submit it to the judgment of the medical profession, with the assurance that, whether favorable or unfavorable, the decision will be just.

ROBERTS BARTHOLOW.

1509 WALNUT STREET, PHILADELPHIA,
September, 1880.

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SPECIAL PATHOLOGY AND THERAPEUTICS.

LOCAL DISEASES.

DISEASES OF THE DIGESTIVE SYSTEM.

TOPOGRAPHY OF THE ABDOMEN: PHYSICAL EXPLORATION.

Regions.—In Figs. 1 and 2 the topography of the abdomen is indicated, according to the usually accepted arrangement. The position of the organs within, relatively to the exterior boundaries of regions, must be definitively known, to make the topographical outlines available for any useful purpose.

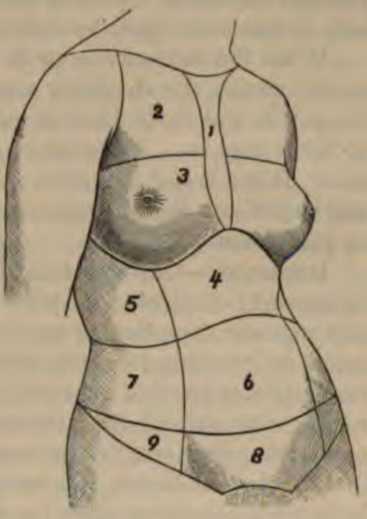

FIG. 1.—THORACIC AND ABDOMINAL REGIONS. *Anterior.*—1, Sternal; 2, Subclavicular; 3, Mammary; 4, Epigastric; 5, Hypochondriac; 6, Umbilical; 7, Lumbar; 8, Hypogastric; 9, Iliac. After Rüdinger.

In the epigastrium (4, Fig. 1) is contained the left lobe of the liver, the pyloric end of the stomach, the first part of the abdominal aorta, the semilunar ganglion and the solar plexus, the pancreas, a part of the transverse colon, the supra-renal bodies, and a portion of each kidney. In the right hypochondrium, we find the liver, gall-bladder, portal vein, the vena cava, the hepatic artery, hepatic and common ducts, the hepatic plexus, the ascending colon, the duodenum, kidney, etc.; and in the left, the stomach, spleen, junction of transverse with descending colon, kidney, etc. In the umbilical and lumbar regions, we find—beginning with the right—the ascending colon, the small intestines, the vena cava, the aorta, the ureters, the mesentery, the splanchnic nerves, the kidneys, the descending colon, etc. In the right iliac region are contained the cæcum, the appendix vermiformis, the iliac arteries and veins, etc. In the hypogastrium are placed the bladder, the ureters, iliac arteries and veins, etc., and in the left iliac region the sigmoid flexure of the colon.

Inspection, palpation, and percussion are the chief modes of examination for ascertaining the state of the organs composing the digestive system, but occasionally auscultation may also be employed. Inspection informs us regarding the condition of the mucous membrane of the lips, cheeks, tongue, tonsils, palate, and pharynx. Inspection by rhinoscopy discloses the condition of the vault of the pharynx, of the posterior nares, and of the orifices of the Eustachian tubes; by laryngoscopy, of the epiglottis, the larynx, and upper trachea. For ordinary purposes an inspection of the throat may be made without the aid of a special illuminating apparatus. The patient is seated opposite the window, the mouth is widely opened, and the tongue is gently steadied—not forcibly held down—by the depressor, handle of a spoon, etc.; now, a deep inspiration is taken, and all parts of the throat, except, of course, the posterior nares and larynx, are well brought into view. Violent attempts to depress and hold the tongue only defeat their object by exciting reflex swallowing, or regurgitation.

When the abdomen is to be examined, the patient should usually be recumbent, the shoulders somewhat elevated, the thighs flexed on the pelvis, and the abdominal muscles relaxed by the voluntary efforts, as far as may be. Examination should be made in the erect position also. It is a useful expedient to outline the regions with a soft pencil, and to indicate by the same means the changes in sonority discovered on percussion.

Inspection.—On *inspection* of the abdomen the following facts may be learned: changes in the color of the skin, and the position of abnormal pigment deposits; prominence or retraction of the walls; movements in breathing (thoracic breathing, abdominal), drawing in during inspiration, instead of outward expansion, jerking or rhythmical respiration; the state of the veins when unduly prominent; exaggerated epigastric pulsations, and presence of abnormal pulsations; distention of the whole abdomen, and undue prominence in particular situations, as in the epigastrium when the stomach is too full, local swellings due to gas, accumulated fæces, enlargement of organs, etc.

Palpation and Percussion.—To the sense of touch in health, the abdomen makes the impression of a soft, flexible, and dough-like material, except where the rectus muscle imparts the sensation of combined firmness and resistance. When the cutaneous reflex is heightened from any cause, or peritonitis exists, the rectus contracting in segments—as it may do—the impression made on the sense of touch may be very confusing.

By palpation we recognize the existence of tender points, or fix the position of painful siezures, enlargement or induration, or changes in the contour of organs, tumors, fecal accumulations, and floating or movable organs. As, however, the application of palpation to the study of the condition of the abdominal organs will, necessarily, be referred to in connection with their diseases, only some general practical suggestions will be made here.

On palpation and percussion of the stomach, the amount of its contents, the area occupied by the organ, the existence of tumors—except those at the cardiac orifice—may be made out. It should not be forgotten that usually, but not invariably, the left lobe of the liver extends across the epigastrium and downward to a varying extent, and that the greater part of the stomach lies under the ribs in the left hypochondrium. The position of the pylorus is not constant. The stomach may, indeed, have a nearly vertical position, and the pylorus extend to, and even reach below, the umbilicus. This is the more apt to happen when the pylorus is weighted by a tumor, which may, in consequence, seem to be attached to some other organ distant from the stomach.

The pulsations of the abdominal aorta in thin subjects, and when abnormally strong, may be readily felt, especially in those persons affected by a depressed state of the sympathetic system. A tumor overlying the abdominal aorta may have a pulsation communicated to it that is not easily distinguished from the pulsations of an aneurism.

In the normal state of that organ, the spleen can not be felt, or defined by percussion with certainty, but when enlarged, especially on a full inspiration, its size and condition may be made out. In doubtful cases, percussion should be practiced when, the patient taking a full inspiration, the spleen is forced down into a position where the area of dullness can be ascertained. When the stomach is enlarged and distended with gases, the left hypochondrium becomes resonant, the percussion note having a highly tympanitic quality. If the spleen be enlarged, and especially if large enough to fill out the space beneath the ribs, the percussion note will be dull or flat. By the character of the percussion-sounds, the dimensions of the stomach can be approximated, in cases of dilatation of the organ.

A distended state of the transverse colon modifies, proportionally, the hypochondriac and epigastric regions; by palpation and percussion it can be ascertained whether the increased dimensions of the bowel be due to fæces or gas, or to both. Impaction occurs, greatly more frequently, in the cæcum, and at the sigmoid flexure, than elsewhere in the large intestine. In some comparatively infrequent instances, the transverse colon has a V-shape, and the apex of the V extends below the umbilicus—a condition of things productive, it may be, of serious consequences, by favoring obstruction, and confusing the diagnosis, by altering the significance of the normal palpation and percussion.

Auscultation.—Auscultation is of far less importance as a means of ascertaining the state of the abdominal organs than inspection and palpation; nevertheless, it may be indispensable; for example—to make diagnosis of an abdominal aneurism, auscultation becomes essential. Only in this way can a pulsating tumor be differentiated from an aneurism.

Although narrowing of the pyloric orifice may not be ascertained by auscultation, yet by careful observation a difference may be detected between the sound caused by the passage of gas through the natural pyloric orifice, and that which is produced by a narrowed outlet. To ascertain this, the stethoscope is placed over the pylorus, and then the stomach is forcibly pressed upon to drive the gas and liquid through into the duodenum. The character of the resulting sound varies with the size of the orifice, and hence the laws of acoustics enable us to approximate, at least, to the degree of stenosis.

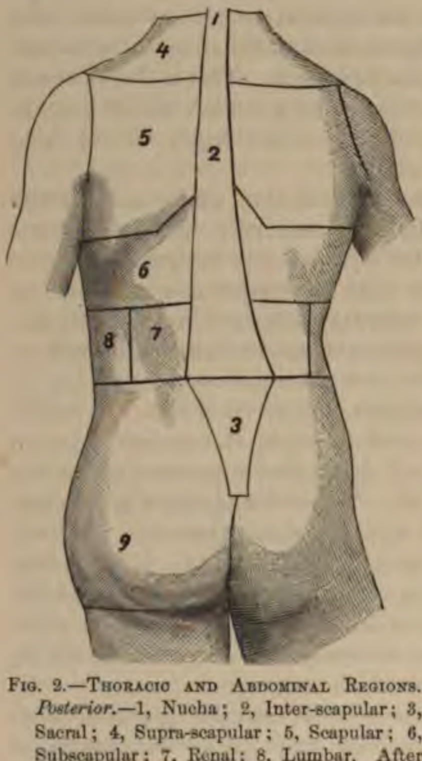

FIG. 2.—THORACIC AND ABDOMINAL REGIONS. *Posterior.*—1, Nucha; 2, Inter-scapular; 3, Sacral; 4, Supra-scapular; 5, Scapular; 6, Subscapular; 7, Renal; 8, Lumbar. After Rüdinger.

Various expedients are now made use of to measure the capacity of the stomach—thus: filled with water after irrigation and then the size can be determined by measuring the fluid.

Also—a sound passed and pushed downward and forward, until the point is felt.

The diagnostic value of the absence of hydrochloric acid in the gastric juice remains a *vexata questio.*

In Fig. 2 the regions of the body on its posterior aspect are given. In the subscapular regions of the right and left sides are contained the organs noted as occupying the hypochondriac regions in front. The renal and lumbar regions contain the ascending and descending colon respectively, the kidneys, the abdominal aorta, and the vena cava, the ureters, the small intestines, etc.

DISEASES OF THE MOUTH, TONGUE, AND PHARYNX.

STOMATITIS.

Definition.—Stomatitis is an inflammation of the buccal mucous membrane. There are various forms of the disease, determined by the seat and character of the lesion—for example: simple, follicular or aphthous, ulcerative, mercurial, and parasitic.

Causes.—Simple stomatitis may be a part of a catarrhal process which involves the mouth, the œsophagus, and the stomach; but more frequently it is caused by local irritants, such as condiments, tobacco, too hot and too cold liquids, etc. The follicular or aphthous form occurs at all ages, but is more common in early life. Children having feeble constitutions depressed by bad hygienic influences are especially liable. Often dependent on gastro-intestinal disorders, it is a frequent complication of prolonged diarrhœa, and more certainly so when the stools have an acid reaction. The ulcerative form is due to all those causes, also, which depress the vital forces—to fatigue, to excesses of all kind, to bad hygiene, to damp and dark habitations, to improper and insufficient food, and to various cachexiæ. Mercurial stomatitis is produced by the systemic action of mercury, in what form or mode soever the metal may be introduced into the organism. It should be remembered that in infancy the mercurial action does not manifest itself in stomatitis, but in an equally injurious toxic action in another form.

Symptoms.—It is almost invariably true of inflammation of a mucous membrane, that the first effect of the process is to arrest secretion of its glandular appendages. The membrane becomes rough and swollen, and of a more or less vivid red color; and the glands, especially those at the base of the tongue, by an increase of their contents, enlarge and become prominent; but the dryness, in a few hours, is succeeded by increased secretion. The fluid now poured out from the surface of the mucous membrane consists of a transparent solution—serum—holding in suspension numberless young cells, cast-off epithelium undergoing fatty metamorphosis, and minute organisms, bacteria, etc., derived from the external air. The exuded fluid tends to accumulate at certain points in the cheeks and on the gums, and on the floor of the mouth. In some places, especially at the mouths of the follicles, superficial erosions are produced by the falling off of the epithelium.

The mouth feels dry and hot at the outset. Considerable pain is experienced at every movement of the lips, tongue, and soft palate, or when hot and cold liquids or irritating solids are introduced into the mouth. Taste is much perverted, or is entirely wanting. The secretion poured out in the mouth excites a subjective taste of foulness, and this is represented, objectively, by an odor of putrefaction, especially when there are carious teeth.

The characteristic of the aphthous form of stomatitis is a fibrinous exudation occurring first in the follicles. The exudation has a grayish or yellowish-white tint, round or oval in shape, and varying in size from the head of a pin to a bean. Subsequently, additions laterally of fibrin bring the isolated deposits in contact, and thus larger patches are produced. The exudation softens in two or three days, the mucous membrane disintegrates, and small ulcers are formed, which cicatrize in a week or two. As a similar process takes place in the skin, in variola, the same terms are used to describe the variations in the aphthous patches; thus they are said to be discrete, coherent, confluent, etc. In infancy the aphthous exudation is arranged somewhat symmetrically, on the veil of the palate, and at the junction of the veil with the bony vault; in adults, the exudation occurring in the follicles assumes a vesicular and pustular character, and attacks the lips, the cheeks, and the point of the tongue.

Considerable suffering attends aphthous stomatitis; the mouth is dry with the initial hyperæmia; but, in a short time, a transparent and viscid secretion streams from the cavity; the ulcers, painful at all times, are exquisitely so when acids, sweets, and sapid substances are ingested, and by the mere movements of the jaws in mastication. The breath is fetid; the sublingual, submaxillary, and parotid glands become swollen and sensitive to pressure. The system at large sympathizes with the local disturbance; and, in children especially, there is more or less fever; disturbances of the digestive organs ensue; the urine becomes scanty and high-colored. It occasionally happens that systemic infection takes place, with all the evidences of the most profound adynamia—the so-called typhoid state. Gangrene of the mucous membrane may then set in, or it may commence in the mouth, inducing an adynamic state. More frequently, aphthæ occur in the mouth as a complication in typhoid or puerperal fever, when gangrene of the mucous membrane may follow.

Muguet is a term applied by the French to designate a form of exudative stomatitis, the special characteristic of which is the occurrence of minute parasitic organisms. The local morbid process is the same as in the other forms of stomatitis; hyperæmia, arrest of, followed by greatly increased secretion; production of new cells and casting off of the epithelium, but without exudation of fibrin. The buccal secretion is usually acid, a condition which favors the growth of parasitic organisms. Atmospheric germs are deposited, and a process of acid fermentation goes on with a correlative growth of microscopic organisms. Whitish masses, looking like curds, are to be seen on the palate, cheeks, tongue, and lips. These masses may remain separate and discrete, or enlarge, cohere, and cover the whole mucous surface. They may also extend into the air-passages, but more frequently into and through the intestinal canal. The extension into the latter organs is not by growth along contiguous surfaces, but by deglutition. In the fauces these curd-like masses interfere with deglutition ; in the larynx with respiration.

The membrane-like exudation of muguet is not truly a membrane, but is a collection of epithelial and mucous corpuscles matted to a mass by the vegetation of oidium albicans. The systemic disturbance produced by it depends on the extent of the patches ; if small in size and discrete, there may be no fever and only restlessness due to the soreness of the mouth ; if confluent, there may be considerable fever. When patches develop in the intestinal canal after the vegetations are swallowed, very decided gastro-intestinal symptoms may be produced. There will be more or less diarrhœa, or the stomach may become excessively irritable, food being rejected as soon as swallowed. The suspension of or serious interruption in the process of alimentation causes an extreme degree of anæmia and impairment of the vital forces with cerebral symptoms, comprehended under the term hydrencephaloid, or spurious hydrocephalus. These cerebral symptoms are frequently confounded with the opposite state—cerebral congestion.

Diagnosis.—The ulcerated form of stomatitis is to be distinguished from syphilitic mucous patches. The distinction rests on the history, the form and duration of the patches, and the presence of concomitant symptoms. In syphilis the ulcers are less sharply defined and contain ashy-gray sloughs closely attached ; they are slow to heal, and appear and disappear ; they are accompanied by other syphilitic lesions, and preceded by a characteristic symptomatology.

The aphthous form of stomatitis, or muguet, may be confounded with diphtheria. The differentiation is arrived at by attention to the following points : In diphtheria the exudation usually begins as a delicate pellicle on the tonsils or veil of the palate ; in muguet as a curd-like or pultaceous mass, on the lips, gums, or cheeks—the former extending forward, the latter backward. The exudation of diphtheria thickens and widens as it develops, and extends into the Eustachian tube, nares, larynx, and to wounded surfaces ; that of muguet is rarely coherent, and extends into the fauces and œsophagus. The exudation of muguet is made up of cast-off epithelium, mucous corpuscles, and the vegetation of oidium albicans ; that of diphtheria, of a true fibrinous material within and upon the epithelium, and an immense quantity of bacteria, which also extend into the neighboring vessels and lymphatics. The odor, the swelling of the cervical lymphatics, the general systemic infection, and the profound adynamia, together with the peculiar sequelæ of diphtheria, separate this malady readily from aphthous stomatitis.

Treatment.—Attention to diet is of the first importance. Acid substances, sweets, and condiments, excite smarting and distress in the process of mastication. In adults ulcerative stomatitis is often due to errors of diet, and such subjects soon learn that acid fruits and vegetables, and those foods which may undergo acid indigestion, will produce a plentiful crop of painful ulcers in the mouth. Obviously, in such cases, the offending articles should be omitted from the diet. The starchy and saccharine substances, owing to their facility for undergoing the acid fermentation, may be equally objectionable. In infants, to avoid the evil effects of acid indigestion, some sodic bicarbonate, or lime-water, is added to the milk. In ulcerative stomatitis, local applications are highly serviceable. The surface of each ulcer should be cleansed and a little pure carbolic acid applied. This produces a momentary smarting, but great relief follows. Salicylate of bismuth, but especially a saturated solution of sodium bicarbonate, have considerable power to allay the irritability and pain. If the local disease be due to gastric disorder, besides regulation of the diet, remedies to allay gastric irritability are necessary : for example, bismuth, minute doses of calomel ($\frac{1}{20}$ to $\frac{1}{12}$), creosote, hydrocyanic acid, etc. In some cases remarkably good results follow the administration of potassium chlorate in large doses—for adults fifteen grains every four hours, and for children proportionately. In aphthous stomatitis the same principles of treatment obtain ; but some attention must be given to the peculiar local conditions. As the extension of the patches is determined, to a large extent, by the growth of the *oidium albicans*, remedies destructive of minute organisms ought to be employed—such as resorcin, salol, iodol, and tannic acid, and a mixture of iodoform and tannin, bismuth, and the salicylate of bismuth, quinine and others, all of these being sprinkled in the form of powder over the seat of morbid action to destroy the parasitic forms and prevent pullulation. A combination of bismuth and carbolic acid is very effective to relieve the extreme irritability of the stomach. Potassium chlorate is equally effective in this as in the ulcerative form. To be successful, it is necessary to administer large doses. Mercurials should never be given in any form, for the destructive ulcerations and the gangrene, which now and then occur, will be attributed to their action.

Mercurial stomatitis will require the same general plan of treatment as the other forms of the disease, with the exception that elimination of the poison must be promoted by the administration of the iodide of potassium.

GLOSSITIS.

Definition.—*Glossitis* is a term that signifies inflammation of the tongue. As usually one half of the organ is involved, the malady is sometimes designated *hemi-glossitis.* When confined to the mucous membrane, it may be entitled superficial or mucous glossitis. When the substance of the tongue is attacked, the disease may be called interstitial or parenchymatous glossitis, according as the interstitial connective tissue or the muscular is the seat of the morbid process.

Causes.—Mucous glossitis is usually caused by the contact of steam, hot liquids, chemical irritants, etc. It may, also, be due to an extension by contiguity of tissue, of stomatitis, and thus constitute a part of a general inflammation of the mucous membrane of the oral cavity. It is usually a secondary disease, arising in the course of various septic maladies, just as parotitis does, which is a more common affection. Among the infectious diseases, of which glossitis may occur as a complication, are erysipelas, pyæmia, puerperal diseases, typhoid fever, etc. Prof. B. Ball maintains that hemiglossitis is a neurosis.

Pathological Anatomy.—In mucous glossitis the anatomical changes are limited to the mucous membrane, and consist in hyperæmia, redness, and swelling, and the epithelium becomes cloudy, granular, and is detached. Especially along the borders of the tongue, and on its dorsal surface, are these changes most pronounced, giving a raw, red, and somewhat glazed appearance to these parts of the organ. In papilliform glossitis the large papillæ of the base of the tongue are swollen by hyperæmia and an accumulation of their contents.

In the deep-seated or parenchymatous glossitis, one half of the tongue is usually involved at the outset. The inflammatory process is thus limited, because of the arrangement of the vessels and the attachment of the muscles along the central tedinous raphé, which opposes a barrier to the extension of the inflammation in this direction. The whole tongue may ultimately become engaged, and is invaded in the more formidable cases. The mucous membrane is swollen, deeply injected, softened, its epithelium detached by a fibrinous exudation. An interstitial exudation separates the muscular elements, which are affected by a granular degeneration in which their striæ disappear; they soften, are completely disassociated, and are finally broken up into a diffluent mass. The interstitial connective tissue, also, participates in the inflammation, the cellular elements undergo multiplication, and, with the wandering leucocytes, form foci of suppuration, which, finally coalescing, constitute a large, purulent collection. The largest accumulation of pus may be at the base of the tongue, and purulent infiltration of the glotto-epiglottic folds may take place. In the subacute form of interstitial glossitis, a hyperplasia of the connective tissue occurs, forming patches of sclerosis; and in the chronic form the new tissue encroaches on the muscular, causing atrophy and degeneration, so that the tongue, or one half, or a smaller portion of it, will be injured and deformed.

Symptoms.—In superficial glossitis there are constantly present a decided heat and irritation of the tongue, and the sense of taste is impaired or lost. Actual pain and an intolerable smarting and burning are experienced in the attempt to masticate, especially if the substance taken into the mouth is acid or pungent. The saliva flows abundantly, and is rather viscid. The sublingual glands appear swollen, and are somewhat tender. The movements of the tongue in speech and deglutition as well as in mastication are painful. The organ is red, raw-looking, sometimes smooth and glazed; the papillæ, in general, are swollen and prominent, especially the circumvallate. In the papilliform glossitis these bodies are much swollen, and are very prominent objects, while the rest of the organ presents a normal appearance. Some heat is experienced in them, and in the act of swallowing they are painful. Very often they cause a feeling of a foreign body lodged on the base of the tongue, and excite repeated efforts of swallowing.

Very different are the symptoms when the body of the tongue is involved in the inflammatory process. The organ enlarges and may become enormous—too large, indeed, for the mouth—and may protrude between the teeth. The swelling, beginning usually on one side, quickly extends to the other, so that ultimately the site of the original mischief is lost in the general tumefaction. Especially does the base of the tongue enlarge, pressing strongly against the roof of the mouth, and, pushing the soft palate into the fauces, forces the epiglottis down on the larynx. With the first swelling the movements of the tongue become stiff and constrained, speech guttural and thick, and swallowing difficult and painful; but when the fauces and larynx are obstructed, swallowing is impossible, speech unintelligible, and even breathing grows more and more difficult. Very great pain in the tongue, throat, and ears, is now experienced; a tough and rather acrid saliva flows from the mouth incessantly; the lymphatic glands of the neck are swollen, often immensely so, and may fill out the whole space from the chin to the sternum; the face is puffy and cyanosed, partly in consequence of the swelling of the cervical glands preventing the return of blood through the jugulars, and partly because the swollen tongue hinders the passage of air into the larynx. So rapid is the progress of the swelling that death may ensue in from twenty-four to forty-eight hours by suffocation, or an increasing stupor announces the onset of carbonic-acid poisoning. The usual constitutional symptoms are present. A more or less decided chill inaugurates the febrile movement; the pulse rises in an adult to 110, 120, or even 140 per minute, and the temperature to 102°, 103°, or in severe cases to 105° Fahr.; the urine is scanty, acid in reaction, sometimes albuminous, and the skin is dry. The tongue is deep red, dry, hot, and of a brawny hardness, except at some point where pus has formed and approaches the surface. At the point of maximum intensity, it may be, when suffocation seems imminent, the case may suddenly improve by the spontaneous evacuation of matter. Then the swelling subsides, the breathing becomes easier, a little liquid may be swallowed, and convalescence is soon established. More or less sloughing of the connective tissue, with consequent deformity on cicatrization, may occur, and gangrene, very rarely. In some instances resolution takes place, without suppuration the swelling slowly subsides from the maximum, and the general state improves correspondingly.

When glossitis comes on in the course of an infectious disease, the swelling develops more slowly than when it is an idiopathic affection, gangrene is more apt to occur, and the general condition is extremely grave.

Course, Duration, and Termination.—In the most acute cases life may be put in jeopardy by the swelling which prevents the access of air in so short a time as twenty-four hours. Chills and high fever—the temperature rising to 104°, 105°, or 106° Fahr.—and sweats will indicate the occurrence of suppuration. Increased difficulty of breathing may be due to an extension of the suppuration, the pus dissecting from the base of the tongue under the glotto-epiglottidean folds, and even to the aryteno-epiglottidean. Spontaneous rupture or an incision permitting escape of matter will afford prompt relief. When resolution takes place without suppuration, the improvement, if it occur, is slow, and the swelling subsides by small degrees from day to day. When glossitis comes on in the course of an infectious malady, which has already taxed the powers of life to their utmost, the additional disease will usually soon determine a fatal result. Sudden death may be produced by an œdema of the glottis, from rupture of an abscess into some of the great vessels, or from paralysis of the heart. The disease may continue several weeks, resolution slowly taking place; or, an abscess discharging favorably, speedy recovery will ensue; or more or less sloughing and loss of substance may occur, a tedious convalescence follow, and the tongue remain impaired in its functions.

Diagnosis.—Glossitis will not be confounded with any other malady, since every step in its development can be watched. Gumma of the tongue may cause some enlargement, but its progress is slow, and is not accompanied by the systemic symptoms of glossitis, and is accompanied by the usual syphilitic manifestations. Hypertrophy of the tongue may be confounded with chronic interstitial glossitis, but the distinction is made by reference to the course of the latter, which leads to induration, usually in patches, and to atrophy of the muscular elements. Secondary swelling of the tongue may result from obstruction to the salivary duct by a calculus, and from inflammation of the sublingual glands. The history of the case and attention to the order in which the parts swelled, the discovery of a salivary calculus in position, and the condition of the tongue, ought to render the differentiation comparatively easy.

Treatment.—The superficial form of glossitis requires the same remedies as stomatitis, or it may be safely permitted to pursue its natural course, a suitable regimen being enforced. The deep-seated form requires more energetic handling. When there is much sthenic reaction, the subject being vigorous, leeches should be applied under the angles of the jaws, or free scarifications of the tongue should be practiced. Water, as hot as can be borne, should be held in the mouth as long and as frequently as possible; or ice may be as freely used, if grateful or more beneficial to the patient. Deep incisions may be necessary to evacuate matter, or if swelling threatens the life by asphyxia. Tracheotomy, or probably intubation of the larynx (method of O'Dwyer), may be required in an extreme case. If swallowing be prevented by the swelling, a flexible tube can be passed into the œsophagus through the nares, and nutritive liquids be thus conveyed into the stomach. Support by suitable aliment is required from the beginning, and the use of alcoholic stimulants must be resorted to as soon as the powers of life flag. At the beginning, if there be much reaction, the arterial sedatives—aconite, digitalis, veratrum viride—may be employed; but quinine, antipyrin, phenacetin, or some other antipyretic may be required to check the formation of pus. At the outset, fifteen to twenty grains of quinine and half a grain of morphine should be given to an adult, and subsequently from three to five grains of quinine and one eighth of morphine, every four hours. If swallowing become difficult, the remedies can be administered in solution by enema, the morphine being suspended if there be any indications of stupor from carbonic-acid poisoning.

TONSILLITIS.

Definition.—By *tonsillitis*, it is intended to express an inflammation of the tonsil. *Quinsy* is commonly used to signify the same disease. Under the term tonsillitis are, however, included several distinct morbid states. Interstitial tonsillitis signifies an inflammation of the connective tissue of the organ; peritonsillitis, of the subjacent connective tissue; and follicular tonsillitis, of the crypts or follicles. The first two are apt to cause suppuration—the last named, to cause ulceration.

Cause.—A relationship has been supposed to exist between the ovaries and tonsils, but it is by no means well defined. Dr. James (London) first mentioned the coincidence of ovarian irritation with inflammation of the tonsils; afterward, Dr. Echeverria, of New York, made the same statement; and recently, Dr. Penrose, of Philadelphia, has connected impotence with the ablation of the tonsils. The rachitic and strumous diatheses and heredity are important factors in the etiology. Those having chronic hypertrophy are peculiarly liable to acute attacks terminating in suppuration. Such are predisposing causes. The most usual exciting causes are climatic changes—exposure to draughts of cold air, the body in a heated and perspiring state, sudden variations in temperature, etc. Follicular tonsillitis with ulceration—ulcerated sore throat—is usually preceded by disorders of digestion, and is apparently dependent on such derangements for its origin. An individual susceptibility to attacks undoubtedly exists, and those who have had them are more liable. Such subjects have seizures every winter, especially if the temperature changes are frequent and the atmosphere damp. Period in life is not without influence. Tonsillitis is rare in infancy and in old age, and occurs most frequently from the second dentition to the thirtieth year. Both sexes are liable, and probably in an equal degree, although males are attacked more frequently because more exposed to the exciting causes.

Pathological Anatomy.—The tonsils consist of a bunch of mucous follicles, held together by connective tissue, and are imbedded in a quantity of fatty and areolar tissue. A morbid process may affect the follicles or crypts, the interstitial tissue, or the subjacent tissue. In acute inflammation, there are intense hyperæmia, redness and swelling of one or both tonsils, and of the palate and uvula. The swelling may be so considerable that the tonsils, meeting in the median line, press aside the pendulum and fill out the cavity of the pharynx. The secretion of the follicles consists of a soft, yellowish, puriform material, which is shown under the microscope to be composed of cast-off epithelium, mucus, and fatty detritus. The connective-tissue corpuscles undergo proliferation, and abundant leucocytes, with some red corpuscles, float in the serum which fills the interstices. Multiplication of the cellular elements finally is sufficient to constitute a purulent collection, which tends to external discharge by gradual softening of the superimposed tissues. When the inflammation occurs in the connective tissue beneath and around the tonsil, the same process takes place, the tonsil participates more or less in the hyperæmia, but it is pushed upward and rather backward by the swelling beneath. When pus forms, it tends to burrow downward to the base of the tongue and under the corresponding pillar of the fauces, which is pushed forward. In the follicular form the hyperæmia is more especially exhibited in the network of vessels surrounding the follicles. The epithelium becomes cloudy from granular degeneration, and is detached. Disintegration of the basement membrane occurs, and thus an ulceration is established. The grayish slough, so long as it remains adherent, bears a superficial resemblance to a diphtheritic membrane, and the two processes are often confounded. Tonsils that have been amputated, leaving behind considerable hypertrophied tissue, are worse in respect to the results of any subsequent inflammation, than the organ in its natural state. The cicatricial tissue is tense, resisting, and hence prevents the escape of pus, or at least impedes its upward extension and ultimate discharge in that direction. The consequence of this state of affairs is the dissection of the pus, downward from the inflamed part, but especially into the base of the tongue.

Symptoms.—A feeling of general *malaise*, aching of the back and limbs, and some chilliness, usually precede the local manifestations in the fauces. These are the well-known disturbances incident to "taking cold." The ulcerative form of tonsillitis is preceded, as a rule, by stomachal distress, by indigestion, acid eructations, etc., and these symptoms are accompanied by an unaccountable languor and debility. The chilliness is followed by fever, which may consist in a very slight elevation of the body-heat (99·5° to 100° Fahr.), or may attain to a more formidable reaction, the temperature rising to 102°, 103°, or even in children to 105° Fahr. Soon after the appearance of these constitutional symptoms, or coincidently with them, uneasiness is felt in the fauces—a sense of heat and irritation—and difficulty, with soreness, is experienced in swallowing. These sensations increase and are constant, and meanwhile paroxysms of acute pain in the fauces, shooting through into the ear, occur spontaneously, or are excited by the effort to swallow. Frequent attempts to swallow are induced by the presence in the fauces of a viscid mucus, which constantly accumulates, and by the enlarging tonsils, which have the effect of an alimentary bolus ready to be grasped by the pharynx. When the attempt to swallow is made, the muscles of the face are contorted, strong efforts of the pharyngeal and cervical muscles are put forth, a burning sensation is felt in the fauces, together with an acute pain which penetrates to the ear, tears come into the eyes, and, with a groan or cry, the act is finally accomplished, a part of the liquid it may be coming back through the nose. In the more severe cases, when both tonsils are affected, the act of deglutition becomes impossible, but usually the severe distress awakened by the effort prevents the attempt, which, if persevered in, may be accomplished in part. Instead of attempting to swallow, the patient tries, by frequent hawking and spitting, to clear the throat of the mucus which accumulates. The voice has a muffled, nasal tone, and words are with difficulty distinguished, or may indeed be entirely unintelligible. Hearing becomes dull, owing to the encroachment of the swollen tonsils on the Eustachian tubes, and noises of various kinds sound in the ears. The breathing is more frequent and less deep than in health, and may, indeed, become labored, inspiration being slow and difficult, if the swelling extend to the aryteno-epiglottic ligaments, or if œdema of the glottis should come on. If the lesions are confined to the tonsils, together with increased frequency and great shallowness of breathing, there are usually paroxysms of suffocative sensations of brief duration and purely subjective.

On inspection, the fauces are seen to be red about the inflamed tonsil, or, if both are affected, the tonsils, palate, and uvula are red and swollen, the redness terminating rather abruptly at the margin of the hard palate. The follicles are swollen. The tonsils, in the beginning of the inflammation, are deeply red, hyperæmic, and prominent, their follicles containing a purulent-looking, semi-solid secretion, which may be mistaken for a membranous exudation. As the swelling increases, the palate and the anterior pillars of the fauces are pushed forward, and the tonsils, if both are affected, meet in the median line. It is in the strumous subject, and in the case of those who have had repeated attacks of the disease, and have acquired a habit, as it were, that both tonsils are simultaneously inflamed. Peritonsillitis, or inflammation of the subjacent connective tissue, is usually—invariably in my experience—unilateral. The swelling lies under and largely in front of the tonsil, and may be felt by the finger as a hard, brawny mass, extending to and embracing the corresponding portion of the base of the tongue. The tonsil, also, participates to some extent in the inflammation, but the formation of pus takes place in the adjacent tissue. This form of tonsillitis usually occurs in those who have suffered amputation of the tonsil. It is tedious, painful, and sometimes dangerous to life, by the pus dissecting down beneath the aryteno-epiglottic folds, or by the sudden occurrence of œdema of the glottis. In cases of tonsillitis of any severity, the neighboring lymphatic glands enlarge, are tender, and the connective tissue about them becomes œdematous. In this way considerable swelling of the neck externally takes place ; when one tonsil only is affected, confined to the corresponding side, and, when both are attacked, the swelling is general. In this way it happens that the movements of the head and neck are constrained and painful. The tongue is usually heavily coated, and the breath fetid, especially in ulcerated tonsils. In this affection also, doubtless owing to the accompanying gastric derangement, there is much debility—out of proportion, certainly, to the local morbid process—and it is not attended by the sthenic reaction which accompanies the other form.

Course, Duration, and Termination.—With the progress of the case much suffering is experienced. But little food or drink, sometimes none, can be taken during the time of maximum swelling, which may continue for two or three days. Rigors, not pronounced, or rather slight chilliness announce the suppuration. On inspection now, a change in the color of the swollen parts may be seen. Instead of a deep redness all over the inflamed area, there may be a circumscribed yellowish spot, at the summit of a prominence in the tonsil, or beneath the tonsil at the base of the anterior pillar of the fauces. Fluctuation may be detected in these places by the finger, passed carefully over the base of the tongue, and two fingers of the other hand resting in the fossa behind the angle of the jaw. This manœuvre is especially adapted to detect fluctuation in cases of peritonsillitis. When suppuration occurs, which is the rule in interstitial and peritonsillitis, the height of the disease is reached in about seven days. Sometimes there is a sudden termination to days and nights of agony by the escape of a large quantity of pus, spontaneously or by incision. The opening may occur in sleep and the pus be swallowed, and the agreeable change in the condition felt on awaking may not be explained. In some cases a number of days may be occupied in the escape of matter by several orifices, the improvement in the patient's state developing gradually. By slow progress in the formation of matter and by its gradual discharge, the duration of the case may extend to two weeks. After the evacuation of pus, rapid improvement takes place. Food can now be swallowed and rest obtained; the fever ceases, and the cavity of the abscess closes. Sometimes resolution takes place without proceeding to suppuration; then the progress of the case is slow, hyperplasia of the connective tissue occurs, and the organ remains permanently enlarged. Successive attacks of inflammation, of a rather subacute type, is the chief factor in the development of chronic hypertrophy. The termination is rather fatal. Œdema of the glottis has caused death. The case of W. J. Wade, as described by the author; also the case of Mr. Rice, Minister to Russia, who was taken when about to sail from New York (May, 1889). A large abscess developing in the course of an acute infectious disease may decide the case unfavorably. Death has been caused mechanically by the epiglottis being forced down by the swelling so as to close the entrance to the larynx, but this is an exceedingly rare event. The duration of follicular tonsillitis is about a week to ten days. The ulcers may be single or multiple, and a large excavation may be produced by the coalescence of several small ones. When the slough is detached, the process of healing goes on rapidly, and the tonsil is more or less changed in form and structure by the new material and its subsequent contraction. Considerable loss of substance may be caused by the sloughing.

Diagnosis.—As every step in the morbid process can be seen, there need be no difficulty in determining the character of the malady. It may not be easy to differentiate between peritonsillitis and interstitial tonsillitis, but, as suppuration is the objective point in both, it is of little consequence to be absolutely accurate. Ulcerative tonsillitis, with slough attached, may be confounded with diphtheria. The distinction is made by observing that the apparent membrane is confined to the tonsil and to its follicles, and does not extend to the palate and other parts.

Treatment.—A saline laxative should inaugurate the treatment unless the bowels are relaxed. Tincture of aconite-root (gtt i—iij) may be given every hour or two for the period preceding pus-formation. Tincture of veratrum viride may be employed in the same way, and with the same limitation, but it is less efficient. Tartar emetic in small doses ($\frac{1}{16}$ gr.) frequently, is also an efficient antipyretic, but, as it is apt to nauseate, the effects are unpleasant.

There can be little doubt now that an antipyretic dose of calomel (grs. iij–v) has a good effect on the morbid process in all directions, and is superior to all other remedies at the onset. Its subsequent use in a similar dose, depends on the state of the fever, the condition of the fauces, and the absence of suppuration. When pus forms, quinine becomes the better agent, and this must also be exhibited in sufficient quantity to make an impression on the local trouble. When an abscess has formed, it is useless to try to change the temperature; then the chief point is to evacuate it as safely and speedily as may be. If the vital powers be depressed, and especially if the pus dissects deeply and causes threatening symptoms, iodide of iron becomes a valuable remedy, and may be given in form of pill or syrup. The author can not approve of the rather reckless administration of potassic chlorate, now so much in vogue, for these reasons: it has no curative effect in these cases, it is a poison to the heart, and it has a very deleterious action on the renal structures. Antipyrin, antifebrin (acetanilid) are more useful than potassic chlorate, and not hurtful in their present or future effects, if used with judgment.

I must caution my readers against the administration of opium or its alkaloids, for when their anodyne and soporific effects are experienced, and deep sleep comes on, fatal œdema of the larynx may occur.

The local treatment may be rather more important than the systemic. If there be much tumefaction of the neck, great relief is afforded by a hot or cold wet pack. A gargle of hot milk and water, used every few minutes, is an efficient means of lessening the inflammation and swelling. Sometimes ice and cold water are more grateful, when they may be used instead. Bicarbonate of sodium, in powder, placed on the base of the tongue, or a solution, in the form of a gargle, gives great relief, it is said, in cases of acute tonsillitis. When suppuration occurs, warm applications are to be preferred. If the tonsils come in contact in the median line and swallowing becomes impossible, great relief to the tension may be effected by scarifications of the surface of the swollen bodies, and the bleeding encouraged by gargling with tepid water. When the formation of pus is rendered certain by the change in the color of the pointing part and by fluctuation, an incision should be made to evacuate the matter.

Guaiacum has long been celebrated for its power to arrest tonsillar inflammation. It should be given early, in scruple doses, for, if suppuration have occurred, no remedy but to secure the discharge of matter will be of any service. Another remedy supposed to have specific powers is ergot. The fluid extract may be given internally and applied locally undiluted. Also a curative action, which has, apparently, an element of specificity, is had from mercury—calomel and hydrargyrum cum creta. These agents must be used with caution, especially if calomel was administered at the outset.

GANGRENE OF THE MOUTH—NOMA.

Causes.—Gangrene is a result in some cases of stomatitis; but these are not, properly speaking, cases of noma, which is a special disease, and occurs as an independent affection. It is a disease of early life—from three to five—and attacks the child of squalid poverty, or those living under the most unfavorable hygienic conditions. It is sometimes an accident of the incautious use of mercurials in unhealthy subjects.

Morbid Anatomy and Symptoms.—The inner face of the cheeks, more usually of the left side, is the favorite site of the gangrenous process. At first a deep-violet or purple spot appears, surmounted by a vesicle full of bloody serum. Softening and destruction of the tissues take place, producing a quantity of sanies and detritus. Large excavations are thus formed, which widen as the destruction proceeds. A horrid stench is emitted from the decomposing mass. The jaws are eroded, the teeth loosened, and the lips invaded. Thromboses close the veins, but the arteries remain permeable; the nerves are stained black, but are not otherwise altered in structure. If a cure is effected, very great deformities may result in the process of cicatrization, and the functions of the parts be seriously impaired.

Usually this disease begins silently and is painless, and hence escapes detection until the appearance of a grayish-black mass attracts attention to the mouth. When fairly inaugurated, the disease extends so rapidly that distinctive symptoms are produced. A pronounced odor of animal decomposition is exhaled with the breath; the lips and cheeks become swollen and œdematous; the sublingual and submaxillary glands enlarge; sanies and bloody saliva, mixed with the gangrenous and decomposing materials cast off from the sloughing ulcer within, are constantly flowing from the mouth. Marbling of the dirty, wax-colored skin with purplish, vein-like lines, and a central dark spot of commencing decomposition, indicate the outward extension of the gangrene to the cheek.

As already indicated, during the first few days of the disease only local symptoms are present; but then auto-infection ensues by reason of the absorption of the gangrenous materials, and an adynamic state is produced. Then the appetite is lost, nausea and vomiting occur, and a fetid diarrhœa supervenes. The strength fails rapidly, the pulse becomes small and weak, and low muttering or merely nocturnal delirium comes on.

Course, Duration, and Termination.—The course and duration of the malady vary with the age, the vigor of constitution, and the hygiene. The gangrenous eschar on the cheek usually forms within the first week, and death may occur by exhaustion at the end of the second week; or the patient may be cut off by an intercurrent malady, notably pneumonia, at an earlier period. Pursuing its ordinary course, without complications, death may result from septicæmia in two weeks. When recovery takes place, the convalescence will be rapid or tedious, according to the amount of tissue to be repaired, and, even after the arrest of the gangrene, the powers of life may be exhausted by the extensive and protracted suppuration. The mortality is great, and ranges from sixty to seventy per cent.

Diagnosis.—Noma is to be distinguished from malignant ulcer, and from ulcerous stomatitis. Malignant ulcer begins on the lip; noma on the mucous membrane within. The former is an ulcer covered with an ash-gray slough; the latter is a mass of blackish, gangrenous, decomposing tissues. The ulcero-membranous stomatitis consists of a number of small, round ulcers, at various points, that do not become gangrenous, and heal readily on appropriate treatment.

Treatment.—Support to the powers of life is the main point, and this includes not only aliment but air-space. Alcoholic stimulants must be used early and freely. Quinine in full doses, and opium cautiously, should be given with the view to arrest the spread of the gangrene, and to prevent septicæmic infection. If administered at an early period, belladonna seems to possess the power to prevent the spread of the gangrene. It is very important to destroy the first sloughing tissue by active caustics, as Vienna paste, chromic acid, zinc chloride, muriatic acid, etc. The caustic must be so applied as to destroy a small extent of surrounding healthy tissue. Other efficient topical measures are resorcin, applied freely by insufflation after the separation of sloughs; turpentine, conjoined with its internal administration, and compound solution of bromine (bromine, ʒ i; potassium bromide, ʒ ij; water, ℥ j).

CATARRHAL INFLAMMATION OF THE NASO-PHARYNGEAL MUCOUS MEMBRANE.

Definition.—The upper pharynx, into which the posterior nares enter, is the seat of this inflammation. It may be acute or chronic.

Causes.—Inflammation of the naso-pharyngeal space is usually a part of an inflammation involving the posterior nares and the lower pharynx. The most prolific cause is taking cold. Next to this is the use of cigarettes, especially if the smoke is inhaled and ejected by the nares; and then comes alcoholic excess, but little less important. Diphtheria, the eruptive fevers, and inflammatory affections of the air-passages, are accompanied by this disease, and it may succeed them.

Pathological Anatomy.—An intense hyperæmia—a vivid redness—is the first change, but in chronic cases the color of the membrane is reddish-brown. As a result of the congestion, hæmorrhagic extravasations may occur. The mucous membrane is swollen, infiltrated, and projecting from the general surface are numerous enlarged follicles. The increase in size of the follicles is due largely to the increase and accumulation of their cellular contents. The pharyngeal tonsils are enlarged from the swelling of the mucous membrane, and the orifices of the Eustachian tubes are changed in form by the same cause, or even obstructed. A quantity of glairy, tenacious mucus is poured out, and coats the surface of the membrane. In chronic cases, the mucous membrane is much altered by the enlarged and tortuous veins, by hæmorrhagic extravasation, and by the hypertrophic enlargements of the follicles. In very old cases the mucous membrane undergoes atrophy. There is also increased secretion; the mucus is mixed with pus, and not unfrequently with blood, and a thick string of muco-pus can often be seen projecting down into the lower pharynx, behind the soft palate. Erosions of the epithelium also take place, and superficial ulcers form.

Symptoms.—There is at first, in acute cases, an unpleasant, stuffy, and dry feeling in the naso-pharyngeal space, followed in a short time by increase of secretion falling into the pharynx or discharging by the anterior nares. There may be some headache and pains in the upper jaws. Breathing through the nose is difficult. The voice is thick and nasal. The symptoms of an acute attack subside in a few days, the secretion changing to a yellow muco-pus from the transparent, glairy mucus which first appeared, breathing through the nose becoming natural, and the voice assuming its normal tone.

In the chronic form, the symptoms succeed to the acute or develop slowly from the causes continuously acting. The posterior nares are more or less obstructed, constantly to a slight extent by the swelling of the mucous membrane, and occasionally very much by accumulation of mucus. Breathing through the nose may be sometimes prevented. The voice is more or less thick and nasal. Pain in the ear may be felt, and dullness of hearing is a common symptom from obstruction of the Eustachian tube. The mucus, hanging down into the lower pharynx, excites frequent attempts to swallow, and causes a feeling of the presence of a foreign body. A disagreeable habit of hawking is induced in this way. In very chronic cases with atrophy of the mucous membrane, secretion ceases, and the membrane has a dry and glazed appearance.

Course, Duration, and Termination.—The course of the acute form is short, and the termination is in health, or in the chronic form. The chronic form is very slow, and is usually regarded of importance only when a thick band of mucus hangs into the lower pharynx, and excites efforts to clear the throat. As a not infrequent cause of deafness it comes under the observation of the aural surgeon. Although curable under appropriate management, the treatment is very protracted. As success in the treatment requires abstention from the two prevalent habits of smoking and drinking spirituous liquors, success will depend on the conduct of the patient very largely. Left to itself, the duration is indefinite.

Treatment.—The first step in the treatment is to free the mucous membrane from the viscid discharge. This is best accomplished by washing out the cavity with the post-nasal syringe, employing a solution of common salt or carbonate of sodium (℈j—℥iv). The syringe is passed behind the veil of the palate, the fluid discharged, when, the patient leaning forward, it escapes into a vessel placed to receive it. So much damage to the ear has resulted from the incautious use of the nasal douche, that the author advises the curved post-nasal syringe for the purpose just indicated. A principle of great importance is to avoid such frequent application of a topical remedy as will add each time something to an existing irritation. To be effective, the remedy must make an impression, but this action should cease before making another impression. Whether solid, liquid, or gaseous remedy be used the same principles should govern.

Of the gaseous or aëriform remedies none is so curative as ethyl-iodide, which can be inhaled without apparatus. It can be vaporized at 100° Fahr., and the simplest appliances will suffice ; the one point is to inhale the vapor slowly and persistently, carrying it through the nose and naso-pharynx until some heat and sense of irritation occur. When the increased discharge of mucus caused by it subsides, another dose can be inhaled. Usually one inhalation a day suffices, or it may be better to inhale it on alternate days, or even twice each day. The author advises a patient and persevering application of the remedy—for his experience justifies him in saying that admirable results may be thus accomplished.

Bromine has been used with success as the author has seen, but its odor is abominable, and its local oxidizing and destructive action is great. If used with caution, i. e., properly diluted with air, its curative power is great.

Many of the best throat specialists find finely divided powders, administered by insufflation, the most effective of the various modes of topical treatment. *Iodoform* of itself, *tannin* of itself, and the two in combination, reduced to the minutest state of subdivision, are the most useful, as a rule. By an insufflator the powder is blown on and over the affected area in sufficient quantity to excite some sense of irritation. The repetition of the application depends on the subsidence of the impression immediately preceding. As iodoform is so disagreeable, iodol may be substituted, and the usual topical applications formerly in vogue the practitioner may advantageously supplant by bismuth, salicylate of bismuth, naphthol, acetanilid, asepsin, and the numerous new antiseptic remedies.

CATARRHAL INFLAMMATION OF THE LOWER PHARYNX.

Pathogeny and Symptoms.—This may be acute or chronic. Both forms arise under precisely the same conditions as the corresponding maladies of the naso-pharyngeal space. The changes in the acute form consist of redness, swelling of the mucous membrane, enlargement of the follicles from accumulation of their contents, and increased secretion, coming on after a very brief dry stage. These anatomical conditions are not limited to the pharynx. In the chronic form, the changes are more decided. The mucous membrane is of a deep reddish-brown, or, in very old cases, grayish. The vessels of the mucous membrane are enlarged and tortuous. The follicles are enlarged and prominent, and have a grayish or reddish-gray color ; there may be considerable development in places of the squamous epithelium, and ulcers, rather shallow than deep, form in various situations. The symptoms are by no means pronounced. Dryness, a sense of heat and irritation, a feeling as if something were adherent to the mucous membrane, much hawking and clearing the throat, are the chief sensations. On inspection of the fauces the mucous membrane is seen to be of a deep, reddish-brown color, thick, coated with a tenacious mucus, and roughened by enlarged follicles. In very old cases the posterior wall of the pharynx is smooth, thin, and glazed, in consequence of atrophic changes succeeding to the inflammatory, and has adherent to it dry masses of mucus, colored by dust.

Treatment.—The principles and the methods of practice advised for the naso-pharyngeal space are equally applicable here.

RETRO-PHARYNGEAL ABSCESS.

Definition.—By this term is meant an accumulation of pus in the submucous connective tissue, posterior to the pharyngeal wall. An abscess may form in the mucous membrane itself—this is entitled pharyngeal abscess.

Causes.—Diseases of the cervical vertebra, of the atlas and axis, as caries, are the principal causes. Large collections are formed in the same situation, from suppuration in the bronchial glands, and in the deep cervical lymphatics—the pus dissecting up under the mucous membrane, and pointing in the pharynx. Again, an abscess may be the result of an inflammation of the loose connective tissue, under the pharyngeal mucous membrane, a disease not infrequent in children before the tenth year.

Symptoms.—The abscess produced by an acute inflammation of the connective tissue is very acute in its course. It begins with chill, high fever, sleeplessness, intense restlessness, and in very young children there may be convulsions. When the abscess results from caries of the vertebræ, its march is slower, and the symptoms of pharyngeal obstruction are the first to call attention to this part. Pain in moving the head is felt, and hence it assumes a fixed position, the cervical muscles being rigid. Then difficulty of swallowing and dyspnœa come on. If digital exploration is then made by passing the index-finger gently over the base of the tongue, a hard, brawny, possibly fluctuating swelling may be detected in the pharynx. The neck will also be much swollen externally, and fluctuation may ultimately be felt under the angle of the jaw. Suppuration is often announced by the occurrence of a chill, and the fever will then assume an intermittent or remittent type, and profuse sweats will occur. The abscess, if not interfered with by art, will discharge spontaneously into the lower pharynx, or externally, or form fistulous communication with the cavity. The author has seen one case in an adult, which extended from the basilar process to the root of the lungs. When spontaneous opening of the abscess takes place, suffocation may be caused by escape of the matter into the larynx. Death may also be caused by the size of the collection, the larynx being occluded, or by secondary disease of the air-passages, or by thrombosis of the transverse sinus, or jugular vein, or even of the carotid artery.

Course, Duration, and Termination.—There are great differences, according to the origin of the abscess, in the course pursued. Those due to caries of the vertebra are slow in development, but fatal in result. The phlegmonous abscess is acute, pursues its course in from five to twenty days or longer, and the danger is determined by the size of the collection, and the direction taken by the pus if not spontaneously evacuated. If not large, the abscess will discharge and heal without danger to life. The large submucous abscess will almost always prove fatal by exhaustion.

Treatment.—Pus should be evacuated at the earliest moment. The powers of life must be sustained by proper aliment and the free use of stimulants. The formation and spread of pus must be limited by the administration of quinine, as far as such a result is possible, and by calcium sulphide, malt extract, the hypophosphites, phosphates, etc.

DISEASES OF THE ŒSOPHAGUS.

CATARRH OF THE ŒSOPHAGUS—ŒSOPHAGITIS.

Causes.—Acute œsophagitis exists only as a part of a morbid process involving the mouth, fauces, and stomach. Typical examples are afforded by the action of irritant poisons and corrosive substances.

The chronic variety is produced by the causes which give rise to the chronic stomatitis. The acute and chronic forms differ so little that they may be considered together. The change in the mucous membrane consists in more or less hyperæmia, especially about the follicles; at first an arrest of secretion, followed by an abundant pouring out of mucus, which in the chronic form is always in excess. Considerable hypertrophic thickening of the mucous membrane occurs in the chronic malady, and in some situations takes on the form of papillary or polypoid-like outgrowths. Coincident thickening of the muscular layer also occurs. Erosions of the mucous membrane, at first superficial, are produced by disintegration and separation of the epithelium, and ulcers are then formed, which may extend to the deeper layers. The greatest diameter of these ulcers is parallel to the long axis of the tube. Ulcers also result from the impaction of foreign bodies; from corrosive liquids; from tubercular deposition, etc. The catarrhal form may be confined to the follicles, when it is called follicular œsophagitis. The follicles are swollen and prominent, partly in consequence of an abnormal accumulation of their contents, and partly in consequence of an hypertrophy and contraction of the adjacent connective tissue. The diseased follicles appear as firm nodules, somewhat conical in shape, projecting above the general surface, and irregularly distributed along the tube. A fibrous or croupous œsophagitis also exists, not as an independent affection, but consisting of an extension downward of an exudation, croupous or diphtheritic, or occurs as a complication in typhus, scarlet fever, small-pox, etc. There is, also, a phlegmonous or purulent inflammation of the œsophagus, which comes on by extension of purulent infiltration of neighboring parts, as in perichondritis of the larynx, by the action of corrosive substances, by lodgment of foreign bodies etc.

Symptoms.—In either acute or chronic form, œsophagitis produces but few symptoms. Pain in swallowing is usually present in the acute form, and may be developed in the chronic cases by the ingestion of hot or rough foods. Pain may be caused by pressure on the tube from without, and by the passage of an œsophageal bougie—a procedure by which we may designate the seat of ulceration, or lesser kinds of irritation, even. When there is severe local disease at any point, as an ulcer, for example, food swallowed descends to that point, excites a sensation of heat and pain, and is then regurgitated by a sudden reflex spasm of the tube. Sometimes mucus or muco-purulent matter will be found adherent to the particles of food. Chronic catarrh is especially characterized by the production of much glairy and tenacious mucus, which rises into the pharynx, causing the sensation of the presence of a foreign body. The attempt to clear the throat of this often excites gagging. These symptoms are, not unfrequently, confounded with those due to corresponding diseases of the throat, especially chronic and follicular catarrh.

Course and Duration.—Simple acute catarrh terminates in a few days. When produced by corrosive liquids, the process of cicatrization will occupy several weeks, and subsequent contractions and strictures may so interfere with nutrition as to cause death by marasmus after many months. The chronic, and especially the follicular, variety may continue unchanged for years.

Treatment.—The management of the various forms of œsophagitis is the same as the corresponding affections of the mucous membrane of the mouth. The topical applications must necessarily be restricted to the agent swallowed.

DYSPHAGIA.

Dysphagia, or difficulty of swallowing, is a symptom of disease, but not a disease itself. It is frequently hysterical, when it is accompanied by other hysterical manifestations, as the globus hystericus, laughing and crying, etc. It may be hypochondriacal, when the patients present the deep dejection, the indifference, and other symptoms of that state. It may be due to stricture, succeeding to injury by steam, corrosive liquids, injuries of various kinds, cicatricial tissue, malignant disease, etc. It may also be due to paralysis of the palate, a sequel of diphtheria. It will be more appropriately considered when these topics are discussed.

STENOSIS OF THE ŒSOPHAGUS.

Causes.—The term stenosis signifies narrowing of the œsophagus, produced in various ways. It may be congenital or acquired : the latter only will be considered here. As regards acquired stenoses, they may be produced by causes acting from without, by compression ; within, by obstruction. As respects those acting from without, we find the lumen of the œsophagus narrowed by tumors, the enlarged thyroid, aneurisms, caseous lymphatics, etc. Obstructions from the interior are caused by foreign bodies lodged, which usually produce acute symptoms, but sometimes remain, lodged in pockets or diverticula, for months or years. Parasitic growths gradually developing may cause stenosis. Fibroid polypi, club-shaped or lobulated, slowly obstruct the canal, and hence cause the symptoms of obstruction very slowly. Strictures are formed by the contraction of cicatrices, or by carcinoma. Cancerous stenoses are more frequent than all the others combined. Their usual seat is the lower third of the canal, and they may involve the whole periphery and a considerable part longitudinally.

Symptoms.—Increasing difficulty in the passage of food, which the patient recognizes at a certain point, is usually the first symptom experienced. Swallowing is successful, but the patient feels a sense of obstruction below, requiring at first repeated attempts at swallowing to overcome; then repeated sips of water, with more swallowing to dislodge the bolus; and, when the obstruction reaches a certain point, regurgitation occurs, not in consequence of an inverted peristalsis, but the mechanical effect of partial compression of a tube containing liquid contents. The position of the obstruction is pretty accurately indicated by the sensations of the patient and by the time when regurgitation takes place. In acute stenosis—from burns, scalds and corrosives—and in chronic carcinoma, when complete obstruction occurs, food is regurgitated as soon as swallowed. The physical signs of stenosis are important. On *inspection* in thin persons, the movement of the bolus may be seen descending to the point of stoppage if high enough up, or the return movement may be discerned. Enlarged lymphatics may be visible at the root of the neck, and the abdomen, especially the hypochondria, may be flattened and retracted, indicating starvation. On *auscultation* the normal œsophageal sound produced by the passage of foods may be heard suddenly arrested at the point of obstruction and passing upward on regurgitation, or various adventitious sounds may be audible, as gurgling, sucking, spluttering, etc., at the point of narrowing. An important symptom is spasm of the glottis, produced by pressure of a growth, especially cancerous, on the recurrent laryngeal nerve. A peculiar cough, sudden paroxysms of difficult breathing, and a toneless voice, are thus caused. Difficulty of breathing may also be due to pressure on the trachea simultaneously with the œsophageal pressure. The most tormenting hunger and thirst arise in the progress of the case, and increase with the increasing difficulty of getting aliment in the stomach; the body emaciates to an extraordinary extent; the mind is incessantly occupied with thoughts of savory viands, and, in the delirium with which the scene closes, the hapless patient is engaged with the most sumptuous repasts.

Diagnosis.—The spasmodic stenosis of the hysterical and hypochondriacal is accompanied by the usual symptoms of these states, and the condition of the patient as to nutrition is not in harmony with the gravity of the local phenomena. Acute stenosis is preceded by the history of injury by scalding or burning, or by the ingestion of corrosive liquids. The question of cancer is to be considered with reference to the age, which is almost always after forty-five, and the development of the disease is marked by a gradually increasing difficulty of swallowing, by marasmus, and the cancerous cachexia. External compression may be produced by enlarged lymphatics, by an hypertrophied thyroid, by mediastinal and cervical tumors; but these can easily be differentiated from all kinds of internal obstruction. An aneurism of the arch of the aorta, by compression of the œsophagus and of the recurrent laryngeal nerve, will cause symptoms not unlike those due to cancer of this tube; but there will be present the signs of aneurism. Diagnosis will in all cases be greatly facilitated by the œsophageal bougie; but this instrument must be used with caution when the canal is much injured, lest perforation be produced by its passage.

Prognosis.—The termination is fatal in a large proportion of cases of stenosis; but excellent results may, sometimes, be obtained by the patient and persistent use of the means of dilatation in cases of stenosis by cicatrices.

Treatment.—As hopeless as the therapeutical management may appear, such results as can be effected by massive doses of iodides, and by galvanism, are so remarkable that I can not fail to urge the trial of such expedients. When the time arrives that the usual treatment makes no impression, ℈j to ℈ij doses of sodium iodide should be administered until active iodism occurs. Galvano-faradism, also, has proved to be highly useful—one electrode on the spine between the scapulæ and the other on the epigastrium.

DILATATIONS OF THE ŒSOPHAGUS.

Causes and Symptoms.—Dilatation is a uniform enlargement of the œsophagus, the whole cylinder usually being involved. A diverticulum is a protrusion from the walls laterally, forming a sac of greater or less extent. Ektasia may be caused by fatty degeneration of the muscular layer, which yields in the act of contracting on the bolus as it descends to the stomach. With increasing dilatation, there is increasing weakness of the muscular layer and consequent dysphagia. Vomiting and regurgitation presently occur; after a while the nutrition fails, and the objective symptoms are similar to those of stenosis, the ultimate result being equally unfortunate. Diverticula may be caused by the lodgment of foreign bodies leading to the formation of pouch-like protrusions. Pressure diverticula are usually situated at or about the junction of the pharynx with the œsophagus, and in the median line, posteriorly, for here the longitudinal muscular fibers are wanting and the pressure is greatest. When fully formed, they are deep pockets, or sacs, of varying length, and may be several inches deep. The first step in their formation is the lodgment of a foreign body; then yielding of the muscular layer of the tube, due to fatty degeneration of the muscular elements; increasing pressure from deposits of food and drink; the final result being a sac extending downward and behind the œsophagus. The mechanical effect of a sac in this situation is to push the tube before it and compress it, so that ultimately the food and drink drop into the sac instead of passing into the stomach, thus causing the symptoms of stenosis. The symptoms, however, develop more slowly than in even the most chronic cases of stenosis. Diverticula occur in the great majority of instances after forty, whence it happens that they are often confounded there is no cachexia, and the symptoms continue for yea ing, variable in size. may often be observed above the leve coid cartilage ; this marks the position of the diverticul The food accumulating here may, by the contraction of t muscles or by the fingers of the patient, be dislodged a regurgitated. The sound enters the sac, but is not tightly by it, as is a stricture, and moves about freely in the cavity. diverticula are found low down, opposite the bifurcation of th and are caused by various inflammatory conditions leading to with the œsophagus. The traction thus caused induces the f of diverticula.

DISEASES OF THE STOMACH.

CLINICAL EXAMINATION.

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ACUTE GASTRITIS.

ses.—The stomach is much affected by atmospherical changes. ustration of this is afforded in the summer and autumnal attacks ous and gastric fevers, so called, induced as they are by the very erable vicissitudes of temperature, the hot days and cool nights autumn. Gastric catarrh occurs at all ages after infancy, and ore frequent in men than in women. The most common causes errors of diet, insufficient mastication of food, swallowing too hot too cold liquids, excessive eating, abuse of ices, condiments, and ces, etc., and especially of alcoholic drinks. Various external in-ences and moral causes affect the digestive functions, as occupation, ercise, sedentary habits, grief, etc.

Pathological Anatomy.—In the simplest cases, the lesions may be so slight as to escape detection; in mild but fully developed cases the changes are about as follows: The mucosa is the seat of a delicate injection occurring in isolated spots, arborescent or generalized to the whole membrane. Usually at or near the cardiac orifice, the injection or hyperæmia is most pronounced. The mucous membrane may be intensely engorged, and covered with a grayish, semi-transparent, and tenacious mucus (Orth). It should not be forgetten that enormous congestion of the stomach may exist in cases of mitral obstruction and regurgitation. The similarity of this to true catarrhal states is rendered the more confusing, because of the quantity of glairy and tenacious mucus found attached to the mucous membrane so firmly as to be washed off with difficulty (Wilks and Moxon). The mucous glands are prominent, and are increased in size above the normal, in consequence of the overgrowth of their contained cells and the hypertrophy of the adjacent connective tissue. In chronic cases, the glands have shrunk (atrophy), or have become cystic, in some situations, because of the pressure produced by the contracting connective tissue. Sometimes the mucous membrane is softened and easily stripped off; then again, it is indurated and much thickened, in consequence of interstitial inflammation. Much confusion has arisen in regard to the term "mamillated," which consists in the formation of numerous small, conical eminences, by the contraction of the sub-forty, whence it happens that they are often confounded with cancer ; there is no cachexia, and the symptoms continue for years. A bulging, variable in size, may often be observed above the level of the cricoid cartilage ; this marks the position of the diverticulum within. The food accumulating here may, by the contraction of the cervical muscles or by the fingers of the patient, be dislodged and is then regurgitated. The sound enters the sac, but is not tightly embraced by it, as is a stricture, and moves about freely in the cavity. *Traction* diverticula are found low down, opposite the bifurcation of the trachea, and are caused by various inflammatory conditions leading to adhesion with the œsophagus. The traction thus caused induces the formation of diverticula.

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When the stomach is dilated the fact can be ascertained by percussion, merely, in some few instances. Filling the organ by syphon with a known measure of fluid is, no doubt, a certain means of arriving at approximate results. The period when certain kinds of vomiting occur, and the amount and character of the vomited matters have high significance.

An examination of the vomited matters to detect the presence of bile, blood, an excess or diminution of acid constituents, etc., should not be neglected. With this object in view instructions to retain the vomit should always be given. The significance of the abnormal contents of the stomach will be found explained in their proper relations.

Forms and Varieties.—The diseases of the stomach are named according to their character and anatomical seat. Inflammation of the stomach is called gastritis, and may occur in the mucous membrane, or in the submucous connective tissue. The mucous variety is known as gastric catarrh, and then consists of two forms—acute and chronic ; the submucous variety is designated phlegmonous or interstitial gastritis, and may also occur in two forms—acute and chronic ; the latter is sometimes called cirrhosis of the stomach. There is also a form of gastritis caused by the ingestion of corrosive and irritant poisons—toxic gastritis. Under the term *embaras gastrique* the French authors describe a light form of gastric catarrh, due to the use of various kinds of indigestible aliment. Severe cases of gastric catarrh, in which, in addition to the ordinary symptoms of indigestion, there is present fever, lasting about a week, have been called gastric fever. Chronic gastric catarrh is only another name for dyspepsia.

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Causes.—The stomach is much affected by atmospherical changes. An illustration of this is afforded in the summer and autumnal attacks of bilious and gastric fevers, so called, induced as they are by the very considerable vicissitudes of temperature, the hot days and cool nights of the autumn. Gastric catarrh occurs at all ages after infancy, and is more frequent in men than in women. The most common causes are errors of diet, insufficient mastication of food, swallowing too hot or too cold liquids, excessive eating, abuse of ices, condiments, and sauces, etc., and especially of alcoholic drinks. Various external influences and moral causes affect the digestive functions, as occupation, exercise, sedentary habits, grief, etc.

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Symptoms.—The initial morbid changes, doubtless, precede the occurrence of objective symptoms. At first, diminution of appetite, labored digestion, nocturnal restlessness, inability to undergo fatigue, supra-orbital headache increased by light, by noises, and by movements of the head, and sometimes accompanied by vertigo, are the symptoms experienced. In some instances, the vertigo is extreme; the patient may fall unconscious for a few seconds, and the vertiginous attacks may be confounded with symptoms of the same kind due to cerebral lesions. Pain is felt at the epigastrium, spontaneous or developed by pressure. The epigastric pain may have a boring character, as if passing through the body straight to the spinal column, or under the angle of the scapulæ. Pain is frequently felt in the left hypochondrium, two inches under the left nipple, or in the immediate vicinage of the apex-beat. The tongue is enlarged, marked laterally by the indentations of the teeth, and is covered over its whole extent with a whitish or a yellowish-white coating. The taste is perverted, indifferent, bitter, or putrid. Especially on rising in the morning is the mouth pasty, sticky, and filled with a bitter-tasting mucus. The appetite is totally lost (anorexia), and the thought of food-taking, especially the appearance of food, excites a sensation of disgust; but considerable thirst is experienced, and drinks, particularly those of an acid character, are eagerly sought after. Nausea is present in varying intensity, and there is usually vomiting, at first consisting of the alimentary substances, then viscid mucus acid and bitter, and finally bilious matters. Bilious vomiting is commonly supposed to indicate special disturbance in the hepatic function, but it really means that by the act of vomiting the gall-bladder is mechanically compressed, and its contents forced through the duodenum into the stomach. The amount of vomiting is usually determined by the amount of food previously taken. If the result of an indigestion, the vomiting is copious; but, under other circumstances, it may occur only occasionally, and then be slight. The sufferings of the patient are always aggravated by errors of diet, and vomiting is certainly provoked by eating indigestible food. A foul odor of the breath, eructations of fetid gas, are due to a failure of digestion, and the occurrence of decompositions, the character of which, and the resulting products, being due to the kind of food undergoing this process. Saccharine and starchy foods become converted into carbonic and acetic acids ; the fatty result in setting free irritating fat acids, and the substances containing sulphur and phosphorus give forth the highly fetid compounds of hydrogen—sulphuretted and phosphuretted hydrogen gases. Acidity and heartburn (pyrosis) are thus caused, and tympanitic distention of the stomach results from the setting free of a great quantity of carbonic-acid gas. The intestinal functions may or may not be disturbed. Usually there is present slight constipation ; yet, if the attack is brought on by the use of indigestible aliment, more or less diarrhœa may occur, and it may be conservative. Mild cases of acute gastric catarrh may not excite the least disturbance in the heat-function, but in young and susceptible subjects there may be some feverishness, the movement being of a remittent type, the maximum temperature rarely exceeding 103° Fahr. When the stomach disturbance is extreme, and the fever persists for several days, the cases are sometimes entitled gastric fever, or they are confounded with remittent fever, especially in malarious regions.

Course and Duration.—The duration of acute catarrh of the stomach is four days to a week. A sudden and rapid cure is sometimes effected by a spontaneous or a forced evacuation, by vomiting, by purging, or by a urinary discharge. The beginning of convalescence is sometimes announced by an eruption of herpes, or by a profuse sweat.

Diagnosis.—Acute gastric catarrh with fever may be confounded with remittent and typhoid fever of the first week, but all doubts will disappear as these maladies develop. Vertigo *a stomacho laeso* (Trousseau) is to be distinguished from similar symptoms due to cerebral hyperæmia. The distinction rests on the age of the subject, the presence or absence of degenerative changes in the vessels, and of the arcus senilis, the history of stomachal troubles, the fugitive character of the symptoms, and the prompt disappearance of the stomach-disease when efficient treatment is instituted.

Treatment.—Simple cases of acute catarrh of the stomach need only abstinence and quiet. If the stomach is much embarrassed, and excesses of the table have been recently committed, or some specially irritating articles of diet have been consumed, free emesis is the most effective treatment. The salts of the metals belonging to the class of emetics are too irritating for this purpose. If vomiting have occurred, it may be encouraged by swallowing large draughts of warm water, which will act as a sedative if the stomach is empty. Weak alkaline mineral waters—as Congress, Hathorn, and Vichy of the Saratoga Springs, and the French Vichy—should be drunk freely. Unhealthy and undigested aliment, which has reached the intestines, should be dislodged by saline laxatives. When there is much biliousness—so called—manifested by a heavily-coated tongue, vertigo, headache frontal and temporal, yellow skin, more or less constipation, urine high-colored, acid, scanty, etc., the mercurial purgatives are held to possess some special curative powers. This is probably true to a limited extent, not because of any action on the liver, but because they increase elimination from the excretory glands of the lower ilium. Podophyllin, iridin, euonymia, and ipecac, are nearly equally effective, but calomel in small doses (one twelfth of a grain) has remarkable sedative effects on an irritable stomach. The officinal effervescing powders, carbonic-acid water, milk, and lime-water, are excellent remedies to check vomiting. A mixture in equal parts of carbolic acid and iodine tincture, of which a drop may be taken, well diluted with water, every few hours, is a most valuable remedy to arrest abnormal fermentations and to check vomiting. A mixture of bismuth and carbolic acid with mucilage, in mint-water, is hardly less efficient. After the more acute symptoms have subsided, the tincture of nux-vomica and the diluted muriatic acid are suitable remedies to improve the tone of the stomach and to restore the appetite.

TOXIC GASTRITIS.

Causes.—As already defined, toxic gastritis is an acute inflammation of the stomach, caused by the ingestion of irritant and corrosive poisons.

Symptoms.—So far as the symptoms are concerned, there is no essential difference in the effects produced by the different irritant and corrosive poisons. Immediately on swallowing, there ensues a deadly nausea, rapid and uncontrollable vomiting, the matters rejected consisting of the contents of the stomach acted on by the poison, shreds of mucous membrane, altered blood-clots, etc. A diagnosis of the form and chemical characteristics of the poison may sometimes be made by observing the character of the stain of the face, lips, and mucous membrane—sulphuric acid causing a friable, blackish eschar; nitric acid a yellowish, leathery eschar; caustic potash spreading widely, softening, and liquefying the tissues. In the stomach, dark-brown, greenish, or black discolorations, with masses of sloughing mucous membrane, are observed. It is rare that the whole mucous membrane of the stomach is uniformly attacked. Usually there is considerable discoloration—uniform, indeed, about the cardia, at the greater curvature, and at the pylorus, leaving large portions untouched. Sometimes only the mucous membrane about the cardia and at the pylorus is attacked (Wilks and Moxon); the extent of the action and the resulting appearances depend on the degree of concentration of the corrosive material. Sometimes the walls of the stomach are perforated, a result more frequently due to the action of alkalies than acids. The mineral poisons—arsenic, the salts of mercury, copper, zinc, nitrate of potash, etc.—produce an intense inflammation with vivid redness and injection. Carbolic acid acts superficially, and hardens and tans the mucous membrane.

Similar results follow the ingestion of certain kinds of food cooked in copper vessels and containing the acetate and other salts of copper, or articles of food that have undergone decomposition, such as sausages, hams, cheese, fish, etc. A violent gastro-enteritis is produced in a few minutes or hours after the swallowing of such aliments. Besides the local there are various systemic symptoms, produced by irritant poisons, either due to the diffusion of the poison or to the reflex disturbance resulting from violent local irritation. Besides the vomiting mentioned above as occurring immediately or very soon after swallowing the irritant, corrosive, or toxic substance, purging sets in, and the same sanies, detritus, and sloughs of the tissues discharged by vomiting pass also by stool. In the case of corrosive sublimate and the metallic salts generally there occur intense colic and tenesmus, and the discharges consist of mucus and blood, and strongly simulate dysentery. Whether or not diffusion of the poison or irritant takes place, there occur great anxiety and depression, a weak, rapid pulse, slow and shallow respiration, cold skin, covered with a cold sweat, retracted features, intense internal heat and thirst, burning in the gullet and fauces—the lips, tongue, cheeks, and fauces, charred, corroded, or softened by the contact of the poison.

Course, Duration, and Termination.—The characteristic feature of toxic gastritis is the suddenness with which symptoms arise, after swallowing some solution or eating certain articles of diet. Soon severe pains in the stomach, violent vomiting, and other symptoms occur, the patient having previously been in good health, it may be. Death may occur from the immediate effects of the poison, from the shock of the injury done to the organs, from the shock and subsequent perforation of the stomach, and peritonitis, combined. Recovery may ensue if the injury done is not too great for repair, the patient passing safely through the period of shock and collapse. The evidences of improvement consist in subsidence of the pain and vomiting, in returning tolerance to food which is bland and unirritating, in the disappearance of all the symptoms of collapse. Surviving the first injury, a fatal result may be subsequently due to the inflammation which follows. The convalescence is necessarily tedious, owing to the very limited surface capable of carrying on the function of digestion. Recovery is apt to be partial, and the nutrition ever after is feeble, owing to the extent of injury—the cicatrices and contraction of the stomach, the stenoses of the orifices of this organ, and of the œsophagus.

Treatment.—Vomiting is to be encouraged by the free use of demulcent drinks. If the toxic agent consists of an acid, as speedily as possible weak alkalies, lime-water, soda, common soap, etc., should be administered. If the offending substance is a caustic alkali, weak acids, common vinegar, diluted acetic acid, etc., should be given. The various mineral salts require their appropriate antidotes: arsenic, dialyzed iron, or hydrated sesquioxide of iron; antimony, vegetable astringents, as green tea, galls, and oak-bark infusion; mercury and copper, albumen and mucilaginous substances; phosphorus, turpentine, magnesia, etc.; carbolic acid, saccharated lime. The stomach-pump should be used not only to remove the poison remaining, but to thoroughly wash out the stomach. To allay pain, and counteract the depression of the powers of life, no agent is comparable to the hypodermatic injection of morphia. Ice should be given freely, and an ice-bag applied to the epigastrium. The morphia must be repeated at regular intervals. No food should be given but a little cold milk at short intervals. Injections of defibrinated blood may be practiced with great advantage as a means of support. The subsequent management depends on the character of the poison, and the nature and extent of the injuries.

PHLEGMONOUS OR INTERSTITIAL GASTRITIS.

Definition.—By this term is meant an inflammation of the walls of the stomach, usually of the submucous layer, and resulting in the formation of an abscess, or in purulent infiltration of the parietes. These abscesses may be single or multiple.

Causes.—Phlegmonous gastritis may occur during the course of pyæmia, or be due to hæmorrhagic infarction or to hepatic obstruction. These abscesses may be acute or chronic.

Symptoms.—The symptomatology of phlegmonous gastritis is exceedingly obscure. The ordinary course is as follows: Usually suddenly, or after an irregular prodromal stage, the patient is seized with epigastric pain, followed by nausea and vomiting, thirst, a weak and irregular pulse, great distention of the abdomen, and diarrhœa. Profound prostration comes on, and finally a low delirium and death. These symptoms do not indicate the nature of the malady.

As it is doubtful whether such cases are ever recognized, the treatment must be conducted on general principles.

CHRONIC GASTRIC CATARRH.

Causes.—The chronic form may succeed to the acute. Heredity exercises an influence in its causation; not in the sense that the disease is directly transmitted, but the type of mucous membrane. Bad hygienic influences of every kind, especially miasmatic influences, and all manner of irregularities of life, are causative. The abuse of spirits, and the habitual consumption of highly-seasoned foods and of condiments and sauces, hasty and insufficient mastication, the frequent use of ices, and overfeeding, are the principal causes of chronic gastric catarrh.

Pathological Anatomy.—The most important changes occur about the pylorus. The evidences of previous hyperæmia exist in a brownish discoloration due to hæmorrhagic extravasation and subsequent changes in the hæmatin, and in more or less varicosity of the vessels. There is constantly present more or less hyperæmia, but not the intense and vivid injection seen in acute catarrh. The abnormal supply of blood to the submucous connective tissue leads to overgrowth (hyperplasia, hypertrophy), and this new material contracting, forces the glands into abnormal prominence, causing that appearance known as mammelonated; but it should not be forgotten that this appearance may be due to a contraction of the organic muscular fiber without the existence of any disease whatever. The gland-tubules also increase in size in consequence of overgrowth of their contents, and they produce a quantity of grayish or yellowish, thick, tenacious mucus, which covers closely and adheres to the surface of the mucous membrane. The overgrowth of connective tissue increases the thickness of the mucous membrane and its resistance to section. Compression of the tubules (glands), by the contracting connective tissue, induces atrophy of their cells. Here and there a gland is obstructed; its secretion having no outlet, accumulates, and a cyst is the ultimate result.

Symptoms.—When a chronic succeeds to an acute catarrh of the stomach, the attacks of the latter become increasingly frequent, and presently it is found that the patient is never free from uneasiness and other painful sensations referable to the stomach. This painful and otherwise disordered digestion is commonly known as dyspepsia.

When chronic catarrh exists the patient is rarely free from some disagreeable sensations, but it is after taking food, chiefly, that he experiences a feeling of weight or fullness, sometimes of pain; but acute pain of a lancinating character, especially when it seems to pass directly through to the back, is more frequently due to neuralgia—gastralgia—or is a symptom of ulcer or of cancer. On the other hand, attacks of neuralgia do sometimes occur in the course of chronic gastric catarrh; but the pain of the latter is more often a sense of soreness diffused over the epigastrium, the greater curvature, and is sometimes felt only in the left hypochondrium. Sometimes this pain may be relieved by pressure; but more usually pressure over the stomach, at any point, develops uneasiness, soreness, or pain. As the pit of the stomach, so called (the triangular space under the xiphoid appendix), is occupied by the left lobe of the liver, and as the stomach lies well up in the left hypochondrium, these facts must be taken into consideration in coming to a conclusion in regard to the seat of pain. Sometimes when the stomach is empty, sometimes when it is full, the pain is greater; sometimes the pain is relieved by taking food, sometimes it is increased thereby. These idiosyncrasies give to each case a peculiar physiognomy. The subjective sense of fullness is confirmed by the objective swelling of the stomachal region. After meals, the discomfort caused by the distention is such that the mere pressure of the clothing gives rise to pain. This feeling of distention is due in part to an irritable state of the mucous membrane, but more especially to the formation of the gases of decomposition. In the normal state, the gastric juice has the power to prevent decomposition, or to arrest it after it has begun; but disease alters these conditions, and food in the stomach may pass through various kinds of fermentation according to its composition—the starchy and saccharine undergoing the acetic, and the fatty, the butyric fermentation. In hasty eating much air may be swallowed—the oxygen is absorbed, and nitrogen and carbonic-acid gas remain. Although formerly denied, it is now admitted that, under some circumstances, air is secreted by the mucous membrane—in certain states of the nervous system, especially. A small quantity of starch or sugar may produce a large volume of carbonic acid, causing great distention, and eructations of a sour liquid (pyrosis). Butyric acid induces a strong sense of heat and burning, gaseous eructations, often highly offensive from the presence of sulphur-compounds with hydrogen. Furthermore, gaseous distention of the stomach affects the muscular movements of the organ, so that the foods are not properly distributed and mixed with the gastric juice. In the regurgitations that ensue, particles of food are brought up, the nature of which is recognized by the patient; it may be acid, bitter, or merely mawkish. Again, by the distention of the stomach, the heart is pushed up and its actions hampered, and, through the intimate nervous communications, palpitation and intermittent pulse and a strongly accentuated second sound are produced. In consequence of the compression of the great venous trunks the return of blood from the head is impeded, and hence the face has a congested, red, and swollen appearance, and the head feels full, and headache and vertigo are present during the time the stomach digestion is going on. In some cases of chronic catarrh, vomiting of food occurs soon after it is swallowed. Later, if vomiting take place, the food is in various stages of digestion, and the vomited matters are highly offensive from the presence of butyric acid and the sulphur-compounds mentioned above. Sometimes the vomited matters will have a pasty or yeast-like appearance, due to the presence of a peculiar fungus—from its fancied resemblance to a wool-pack called *sarcina ventriculi.* Vomiting is not constant nor regular, and in many cases occurs only when improper food has been taken. On the other hand, morning vomiting of topers is a constant and ordinary condition in these subjects. As soon as they arise in the morning a feeling of qualmishness comes on, and they strain a great deal to bring up some acid, glairy, tough mucus, or a quantity of rather thin, frothy, watery fluid mixed with air, and alkaline or neutral in reaction, and consisting chiefly of saliva swallowed during sleep. The appetite is usually diminished, or it may be capricious, and rarely excessive (bulimia). Usually but little food in the stomach develops a sense of satiety. Certain kinds of food, by the mere sight or remembrance of them, excite disgust and nausea ; and, as a rule, the animal foods are disliked, and acid fruits and fresh vegetables are craved. The saliva is usually increased in amount ; the tongue is pointed, red at the tip and edges, and the mucous membrane is glazed ; the large papillæ at the base are swollen and tumefied, and there is present more or less follicular pharyngitis. The intestinal functions rarely continue undisturbed ; constipation and flatulence are usually present, and the constipation alternates with diarrhœa. An extension of the catarrhal process from the duodenum to the ductus communis and the smaller ducts causes more or less swelling and obstruction and, consequently, jaundice. The nutrition of the body is impaired by chronic gastric catarrh ; the strength is lessened, and the subcutaneous fat diminishes ; the muscles lose in volume and decline in power, and the various functions are performed with less energy and efficiency. This depression in the functions is especially marked in the psychical sphere, where it manifests itself in melancholy and hypochondria, the patient being solely occupied with his own miseries, and especially with those sensations and feelings belonging to his own state. The peculiar troubles of this mental state are enhanced by the headache, the vertigo, and the other cerebral symptoms which accompany stomachal diseases.

Diagnosis.—The coexistence of the cerebral symptoms just mentioned with those of chronic gastric catarrh may greatly embarrass the diagnosis, but usually the differentiation may be made by reference to the history of the case, the extended duration of the gastric symptoms, which is incompatible with the fact of a cerebral malady, and the absence of concomitant evidences of disease of the nervous centers. Ulcer of the stomach may be confounded with chronic gastric catarrh, but the diagnosis may be made by attention to the following points : In ulcer, there is in front a fixed point of pain, posteriorly a corresponding painful spot ; there is no diffused soreness ; there is acute pain as well as soreness ; the pain is aggravated by pressure, by the ingestion of solids and liquids, especially if hot or cold; there is vomiting of blood. In cancer, there is pain, acute or lancinating or burning, when the stomach is empty or full; vomiting of food, of glairy mucus tinged with blood, and vomiting of black blood; rapid and continuous emaciation; a peculiar icteroid, earthy hue; a tumor, hard or with nodosities; enlargement of external glands (the sub-clavicular).

Course and Duration.—The duration of chronic gastric catarrh is very variable; it may last months or years, now better, now worse, depending on the measures, or the neglect of them, employed for relief. Readily enough cured, if the patient will submit to the regimen necessary, it becomes exceedingly difficult if the causes which produced it continue in operation. Catarrh may terminate in ulcer, or it may lead to stenosis of the pylorus.

Treatment.—The treatment of chronic gastric catarrh due to hepatic obstruction, to valvular disease of the heart, and to albuminuria, belongs to the management of these diseases respectively, and need not be considered here.

Regulation of the diet is of the first consequence in all stomach diseases. All articles that disagree, whether owing to their nature or to idiosyncrasy, should be omitted. As acetic- and butyric-acid fermentations play so important a part in stomach derangements, it is highly important to exclude from the diet those substances the decomposition of which results in the formation of these acids. These articles of diet are the saccharine, the starchy, and the fatty. The mucus acts as a ferment, and these decomposing substances enact the same *rôle*, so that, when the starches, sugars, and fats reach the stomach, the fermentation begins. To exclude these articles, then, is the first step toward a cure. In lieu of these components of the diet, so important to most persons, the succulent vegetables, as lettuce, celery, spinach, cauliflower, tomatoes, etc., should be substituted. The materials for continuing the fermentations, consisting of mucus and the remains of previous fermentation, must be removed from the cavity if a continuance of the disorder is to be prevented. This can be accomplished in several ways: by the use of an absolute diet until the organ has freed itself of its decomposing contents; by the administration of emetics and laxatives; by washing out the organ with the stomach-pump; and, lastly, by the employment of certain medicines. A curative measure of the highest importance is the "skim-milk cure." This consists in the exclusive use of milk for food until the stomach is freed from the materials of fermentation, and has had sufficient rest to recover. The milk is taken in the quantity of four ounces (about) every three hours, day and night, when awake, and for a period of time determined by the cessation of the symptoms for which it was prescribed.

During this time nothing whatever is swallowed, except a laxative to relieve the constipation, or medicine for other purposes; but no medicines should be administered during a course of the milk-cure, unless imperatively demanded. When, after a few weeks, or a month or two, the symptoms of gastric catarrh have subsided, then some additions to the diet may be made, very gradually, consisting at first of a little stale white bread, then rice, then a soft-boiled egg, and so on, gradually, until a suitable diet is constructed.

An emetic, occasionally, is highly useful to empty the stomach of decomposing materials, and to prepare a clean surface for the action of medicaments. Saline laxatives may be employed for the same purpose. An occasional Seidlitz powder; now and then a drachm or two of Epsom salts in the early morning, or the Saratoga waters, or Püllna, or Friedrichshall, etc., are appropriate for this purpose. When there is much biliary derangement, phosphate of soda is highly serviceable. Still more effective for cleansing the stomach is the stomach-pump, or the fountain-syringe used as a siphon. With this instrument the cavity may be thoroughly washed out with tepid water, solution of common salt, solution of potassic chlorate, solution of salicylic acid, etc. As the effects are mechanical, chiefly, and are due to mere washing of the mucous membrane, it usually suffices to employ warm water. In severe cases the irrigation of the stomach may be practiced daily. This manœuvre is readily executed by the use of Debove's tube—a flexible rubber tube about five feet in length, three eighths of an inch in diameter, and having an expanded bell-shaped orifice, into which, as into a funnel, fluids can be poured. This tube has a distinct mark, nineteen inches from its distal extremity, to indicate the distance from the teeth to the cavity of the stomach. As the tube is passed into the œsophagus, the patient is told to swallow repeatedly. When the stomach is reached the fluid intended for irrigation is poured into the tube, the orifice held well up until the organ is full, when the tube is turned downward and made to act as a siphon.

Arsenic is a remedy of the first importance in the treatment of catarrh of the stomach. It is best administered in the form of Fowler's solution, one or two drops three times a day before meals, and it should be continued for a month or more. Next to arsenic, the oxide of silver is to be commended, in pill form, one half to one grain, three times a day, also administered on an empty stomach; but, as argyria may follow its prolonged use, it should not be given for a longer time than one month. When there is much acidity, it may be checked by the mineral acids, notably the muriatic, given before meals. This practice is based on the principle that acids before meals prevent the outward diffusion of those constituents of the blood which contribute to form the acid gastric juice. Alkalies, although they afford relief, do not effect a cure, except in those cases of acidity of a temporary character due to fermentation of starchy and saccharine food, and accompanied by catarrh of the bile-ducts, and then the alkali most effective is the phosphate of soda. When acid is deficient, good results may be obtained by the use of alkalies before meals, on the well-recognized principle that an alkaline fluid in the stomach will favor the diffusion from the blood of its acid-forming constituents. When abnormal fermentations constitute the chief or only source of discomfort, the most serviceable remedy is carbolic acid, alone or in combination with bismuth. Gaseous eructations are best relieved by the same means. Freshly-burned charcoal, finely divided, is a good remedy, though only palliative, acting merely as an absorbent. After suitable treatment for the relief of the local condition, tincture of nux vomica is an excellent stomachic, especially adapted to the chronic catarrh of spirit-drinkers. The bitters in general, with or without the mineral acids, are applicable under the same conditions. It should never be forgotten that all special stimulants to the gastric mucous membrane are injurious, and should never be employed until the morbid state is removed. To employ them without proper regulation of the diet is simply to add another source of irritation. It can not be too strongly impressed on the reader that rest, which is essential to the treatment of any diseased organ, is equally necessary to the stomach when it is suffering; but, as some aliment is absolutely necessary to life, the stomach can never be put into a state of complete repose. Hence the need of a most careful regulation of the diet, so that the condition of rest may be, as nearly as possible, attained. Alimentation by the rectum is a precious resource under these circumstances. Finely-powdered beef, meat-extracts, peptonized foods, eggs, etc., to which the digestive ferments are added, can be administered by the rectum. Defibrinated blood is, no doubt, in the greatest proportion of cases, the most valuable aliment for rectal use.*

ATONIC DYSPEPSIA.

Definition.—By atonic dyspepsia is meant a form of indigestion due to a depressed state of the stomach. It is that form of functional derangement usually called dyspepsia.

Causes.—It is often inherited. It is a disease of advanced life, and is then accompanied by those senile changes belonging to that period, and is a consequence of them. It is a symptom in depressed states of the system generally, as, for example, in exhausting discharges, as hæmorrhages, leucorrhœa, profuse suppuration, etc. It is produced by all those circumstances comprehended under the term bad hygiene. The most influential factors are improper and excessive alimentation, and severe mental and physical exertion immediately after eating.

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* Full particulars are given in the author's "Treatise on Materia Medica and Therapeutics," fifth edition.

Morbid Anatomy.—This malady has not, properly speaking, a morbid anatomy: besides anæmia and deficient secretion, there are no changes. Various alterations have been noted, as atrophy of the tubules, fatty degeneration, increase of the connective tissue, etc. But these changes belong to other states, of which atonic dyspepsia is merely a symptom.

Symptoms.—A sense of weight and uneasiness, lasting throughout the process of digestion, suspended for a short period when food is taken, is usually the initial symptom. A feeling as if a foreign body were lodged behind the sternum, or higher up in the œsophagus, often with a sense of oppression or dyspnœa, is frequently experienced. Acute pain is rarely felt, but there is usually some flatulent colic, and pressure fails to develop pain, but rather affords relief to uneasy sensations. Digestion is impaired in respect to all classes of foods, farinaceous, saccharine, and fatty; and hence, during the process of digestion, flatulence from the formation of carbonic acid and eructation of rancid fats are frequently present. More or less intestinal disturbance accompanies the stomach symptoms, and constipation almost always occurs. The appetite is usually feeble, and the disinclination for food includes all the varieties. There is little thirst, and the ingestion of fluid gives rise to distress. The tongue is too large, and is marked along its borders by the teeth, and is at the same time pale and flabby. The mucous membrane of the mouth is also pale and the gums are soft and spongy; the tonsils are apt to be enlarged, the uvula relaxed, the voice husky, and there is frequent clearing of the throat. The bodily condition generally is that of depression; the pulse is weak, excitable, and easily compressed; palpitation occurs quickly on exertion and frequently without effort of any kind; intermission of the pulse-beat is by no means uncommon, and attacks of pseudo angina pectoris take place, and they may have an alarming character, especially in persons after middle age affected with degenerative changes in the vascular system. Flatulent distention of the abdomen induces oppression of the chest, but dyspnœa may occur without such cause, being due to a nervous state. The skin is usually pallid and earthy, moist and clammy, and the extremities cold. The urine is pale, of low specific gravity, and loaded with the phosphates. The mental condition is in harmony with the general state —that is, depressed. There is great inaptitude for mental exertion, an impaired state of the memory and attention, and irritability of temper. Drowsiness supervenes after eating, while sleep at night is restless and unrefreshing.

Diagnosis.—Atonic dyspepsia differs from chronic gastric catarrh in respect to the amount of pain, vomiting, and tenderness on pressure, which are less, and the depression which is greater, in the former than in the latter.

Treatment.—In this as in other stomach disorders, the first step consists in regulation of the diet. It is useful to commence the dietetic management by the milk-cure. Next, as rapidly as possible, nutritious but easily digested articles must be added. As the digestive powers are feeble, food must be given in small quantity but frequently. As the foods disagree, irrespective of their quality, obviously quantity and frequency of ingestion are the points to be considered. As the powers of digestion are depressed, the special aids to this function are indicated: pepsin, lacto-pepsin, in combination with muriatic acid; pepsin and bismuth with aromatic powder; tincture of nux vomica, strychnine, and the bitters, especially calumba, with or without muriatic acid; the mild chalybeates, as massa ferri carb., the citrate, malate, or tartrate of iron, etc., are the most appropriate of the medicinal agents. A small quantity of acid wine at dinner is a good stimulant to the digestive function. A moderate dose of whisky, taken before meals, is a capital remedy to promote the appetite and the digestion; but it is a dangerous remedy, for it so overcomes the feeling of depression as to be very grateful, and there is therefore a constant temptation to repeat the dose. As, in these cases, there is usually more or less mental depression, change of scene, travel, and agreeable occupation, contribute materially to the cure.

GASTRALGIA.

Definition.—Gastralgia is a painful state of the sensory nerves of the stomach, induced by various sources of irritation, and free from fever.

Causes.—Doubtless the chief factor is a peculiar state of the nervous system, the neurotic temperament, so called, or the nervous state or hysteria. This condition of the nerves existing, various substances, which under ordinary circumstances would not excite the least distress, now cause severe pain. It is highly probable that the abuse of tea and coffee has no little influence in causing the disease.

Symptoms.—The characteristic symptom of gastralgia is the occurrence of severe paroxysmal pain, felt in greatest intensity at or about the epigastrium, and radiating thence upward over the chest and downward through the abdomen. The pain also is felt in the back, and seems to pierce through the body, and it shoots upward to the shoulders. The pain is not increased but diminished by pressure, and the patient instinctively lies or presses firmly on the abdomen, or demands to be rubbed or beaten on the back. In the severest cases, the pain is so excessive as to produce profound prostration; the pulse is small, rapid, and weak, the surface is cold and covered with a cold sweat, and the features are shrunken. In almost all cases, the action of the heart is disturbed, owing to the intimate nervous communications between the two organs; the pulse is small and weak or intermitting. The duration of the attacks is very variable, lasting for a few hours, for a day or two, or continuing for months with intermissions or remissions. Usually the attacks are of short duration, and terminate with eructations of gas, with vomiting, or the more acute pain subsides, leaving a sense of soreness, and occasional lighter pains, which may continue for several days. The attacks may be regularly intermittent, in cases of uterine disease, and when caused by malaria. During the interval, the function of digestion may proceed undisturbed, and the nutrition of the body continue at the normal. Various disorders of the nervous system are usually present, as—palpitations, *migraine*, hysterical phenomena, notably the globus, etc. In males, hypochondria, associated with oxaluria, is not infrequent.

Course and Duration.—Gastralgia is an essentially chronic malady, in that the attacks are prone to return from time to time, and the associated disorders continue in the interim to plague the patient. Those cases dependent on malaria, or on the presence of indigestible food, may be cured with comparative facility, but the ordinary cases are not readily cured. Notwithstanding the obstinacy of these cases, gastralgia is not dangerous to life.

Diagnosis.—Gastralgia is to be differentiated from myalgia affecting the abdominal muscles, intercostal neuralgia, hepatalgia, neuralgia of the solar plexus, ulcer of the stomach, and cancer. In myalgia the pain is restricted to the affected muscles, and has not the acute and lancinating character of gastralgia, and is unaccompanied by nausea and vomiting. As respects intercostal neuralgia, it is to be noted that the pain is in the left hypochondrium, that painful points can be developed by pressure in the course of the nerve-trunk, and at the spine, and that this affection is unaccompanied by nausea and vomiting. To separate gastralgia from neuralgia of the solar plexus is in some cases extremely difficult; but attention to the following points may prevent error: in gastralgia, there is a history of previous stomachal disorders; in neuralgia of the solar plexus, the inhibition of the heart's action is greater, and the systemic depression is more profound. Hepatalgia and hepatic colic are to be separated by the situation of the pain in the right hypochondrium, by the tenderness in the region of the gall-bladder, by the symptomatic fever, and by the jaundice. From cancer, gastralgia is differentiated by the age of the subject, by the character of the vomited matters, the persistence of the pain, the cachexia, the emaciation, and the tumor; from ulcer, by the fixedness of the pain, its constant presence with soreness, the vomiting of blood, etc.

Treatment.—During a paroxysm, the first point is the relief of pain. This may be most effectively and promptly accomplished by the hypodermatic injection of morphine, and frequently so small a dose as one twelfth of a grain suffices. As there is always danger of opium-habit in these cases, this fascinating remedy must be used with caution. Opium or morphine is frequently prescribed with bismuth and aromatic powder. Morphine is also used endermically—that is, applied to a blistered surface, about a square inch of the skin being denuded. By enema is an efficient mode of administering the anodyne. When, from any cause, morphine can not be given, the pain, as also the nausea and vomiting, may be arrested by creosote or carbolic acid. This remedy may also be administered with bismuth in an emulsion—a combination of the most efficient kind. Equal parts of tincture of iodine and carbolic acid, of which a drop may be administered every hour in a little cold water, is a most valuable agent, not only for the relief of pain, but to stop the vomiting. In many cases nitro-glycerin has an excellent effect. The centesimal solution is the most appropriate form for administration—one drop every half-hour being given until relief or the characteristic effects are produced. Arsenic (one drop of Fowler's solution) and opium (two to five drops of the tincture) are not unfrequently highly serviceable for the relief of the paroxysms, but they are more generally useful for the accompanying condition of the mucous membrane, and the end organs of the nerves of the stomach. There is no remedy so constantly curative of the local causes of the attacks, and so efficient in preventing their return, as arsenic. For the condition of things between the attacks, next to arsenic, stand the oxide and nitrate of silver. For the strictly intermittent cases, occurring at a fixed hour, quinine is invaluable; but the author has seen cases which were not removed by quinine, but ceased promptly when salicylic acid was administered. When attacks of gastralgia are due to indigestible food, the first duty is to empty the stomach. If vomiting is going on, it may be encouraged by large draughts of warm water; if vomiting has not occurred, it should be induced by an emetic, preferably by apomorphine administered hypodermatically, to avoid irritation of the stomach. If acid and fermenting materials remain to keep up the disturbance, they should be removed by irrigation of the stomach, or by mild laxatives of the saline and antacid character. It is generally better to remove the contents of the stomach before administering anodynes. The subjects of gastralgia are usually of the nervous, hysterical, and hypochondriacal type, and require chalybeate and supporting remedies. As the stomach in such subjects is easily offended, only the milder preparations of iron can be given—such as the carbonate, the citrate, lactate, etc.; but, in some persons of a habit feeble and relaxed, the more astringent preparations do better—for example, the sulphate and the chloride. Excellent results are often obtained from the use of the mineral acids, notably the muriate, and especially when administered conjointly with the tincture of nux vomica (Fox). The long-continued use of arsenic in a small dose—one drop *ter in die* of Fowler's solution—is more effective, according to the author's experience, than any remedy mentioned. As attacks of gastralgia are, very frequently at least, excited by indigestible food, it is highly important to regulate the diet. Furthermore, in these subjects the digestion has been enfeebled by the depressed state of the nervous system. The best results are therefore obtained by a careful regulation of the hours of eating, the quality of the food, and the mental and bodily exercise. In most cases, probably, the treatment should be begun by the milk-cure, and subsequently a dietary should be constructed suitable to the needs of individual cases. In some instances, the frequent use of a small amount of food is more serviceable than the taking of ordinary meals. When the digestion is feeble merely, pepsin and lactic or muriatic acids are most useful. When acidity and heartburn exist, due to the fermentation of the starches and sugars, the mineral acids must not be given after meals, but before, for chemical reasons already explained.

ULCER OF THE STOMACH.

Definition.—By the term ulcer is meant a solution of continuity involving the mucous membrane and one or more of the layers of which the wall of the stomach is composed, with defined margins having a greater thickness than the adjacent healthy tissues. Symptomatically, the stomach-ulcer is characterized by pain, disorders of digestion, and vomiting of blood.

Causes.—Ulcer of the stomach is a comparatively common disease, and is found to exist in five per cent. of the deaths from all causes. It is present in proportionately greater numbers after thirty-five, because it is an essentially chronic malady; but it is, really, more frequent in youth and middle life, from fifteen to thirty, and it is comparatively often seen in housemaids of twenty—an age, too, at which rupture occurs in greater proportion than at any other. It is probable also that women are more subject to the disease than men, and that rupture occurs more frequently in the former than in the latter. The most influential factors in its pathogeny are, variation in the tonus of the gastric vessels and mechanical arrest of the circulation at the point where the ulcer forms (thrombosis, embolism). There is usually, in these cases, disease of the arterial tunics (atheroma and endarteritis), which finally causes coagulation of the blood and arrest of the blood-stream in a nutritious artery ; obstruction of the portal circulation may induce thrombosis, hæmorrhagic infiltration, etc. The result of a sudden and severe diminution in the amount of blood passing to a part, or of its entire arrest, is to diminish the alkalescence of the deeper layers of the mucous membrane, and to permit the corrosive and solvent action of the gastric juice. It has long been recognized that amenorrhœa, anæmia, chlorosis, the puerperal state, prolonged lactation, and tuberculosis, are also etiological factors, and probably because, in these states, a necrotic process is readily induced, under favorable local conditions.

Irritation of certain parts of the brain is followed by ecchymoses and erosions of the mucous membrane of the stomach. Burns of the chest and abdomen sometimes cause ulceration of the duodenum. A peculiar state of the nervous system must, therefore, be regarded as one of the causes of this disease.

Pathological Anatomy.—Ulcers corresponding in every respect to those of the stomach are found rarely at the lower part of the œsophagus, at the first part of the duodenum (associated with burns on the surface ?), and in the cæcum, as the author has shown. In twenty per cent. of the cases of stomach-ulcer, they are multiple, but rarely as many as five existing at one time ; in eighty per cent. of the cases, the ulcer is solitary. Not all parts of the stomach are equally liable to the ulcerative process. In four fifths of all cases the ulcer or ulcers are found on the posterior wall, the lesser curvature, and about the pylorus. In size they vary greatly, according to age, and probably, according to their nature ; but they are not smaller than a dime, and never attain greater dimensions than six inches by three. In shape they are round or oval, more frequently round. So great is the difference in size, quality, and appearance between the so-called acute perforating ulcer and the round, indurated, and chronic ulcer, that it is difficult to realize that they are merely stages of the same process. The former is about the size of a dime, or shilling-piece, is round and has smooth edges without induration and increased thickness, frequently covered with a clot or containing a mass of slough adherent, and extending in depth to the submucous connective tissue. Ulcers of this description are usually found in young subjects—housemaids notably—have a great tendency to perforate, and are not unfrequently produced by obstruction to the portal circulation (hæmorrhagic erosion, thrombosis, etc.). The latter or chronic form is large in size, having walls of great thickness and indurated, composed of connective and granulation tissue deposited at various times, giving to it a stratified appearance. After many years, such an ulcer presents a crater-like aspect, with shelving sides, and terminates by a small apex in muscular, sub-muscular, or peritoneal layer, or in a perforation. The connective and granulation tissue, of which the crater-like internal surface is composed, is also deposited at the base, and in this way perforation may be prevented. Facts are wanting to demonstrate an intermediate or transition stage between the two forms of stomach-ulcer. In the course of development of the chronic ulcer, the anatomical elements of the mucous membrane, including the tubular glands, are destroyed, and in rare instances villous or polypoid growths appear in the neighborhood of the new formation. In very rare instances the mucous membrane may be largely preserved, and the ulcerative action excavate a cavity beneath. Several small ulcers may coalesce, unite in their long diameters, and thus form an oval excavation along the lesser curvature, or make a girdle around the pylorus. Ulcers of the stomach tend to spontaneous cure. In many instances of death from other causes, ulcers, either healing or cicatrized, have been found when no symptoms had existed during life, in any sense indicative of their presence. In the process of cicatrization, if the ulceration has not extended beyond the muscular layer, the repair is by union of granulations, and the cicatrix forms a puckered depression. When there is more extensive loss of substance, involving all but the peritoneal layer, there is very great contraction, and a large cicatrix with radiating lines of thickened connective tissue remains. The peritoneal surface is drawn in, giving to that membrane a puckered appearance. If the ulcer had been large, oblong, and formed by the coalescence of several smaller ulcers, and situated near the pylorus, narrowing of that orifice, and consequent dilatation of the rest of the organ, were necessary results. Sometimes the base of the ulcer forms adhesions to neighboring organs in the process of cicatrization, causing ever afterward serious interference with the movements of the stomach, and therefore impairing its functions. Secondary cavities are, occasionally, formed by a local peritonitis arising from perforation, the contents of the stomach being prevented escaping into the general cavity of the peritonæum by a limiting inflammation which secures firm adhesion to neighboring organs, to the omentum, pancreas, liver, the adjacent lymphatics, the transverse colon, the kidneys, the diaphragm, and the abdominal walls. If cicatrization take place after these attachments have formed to adjacent organs, they are embraced in the cicatricial tissue, and very great deformity, with serious impairment of function, result. Unfortunately, these conservative adhesions are not always formed: the ulcerative action may continue, cavities be created in the manner already indicated, or communications be established between the stomach and colon, or a fistulous sinus be made through the walls of the abdomen externally, or the diaphragm be perforated and the thoracic cavity entered. When perforation takes place, there being no limiting inflammation, nor adhesion to adjacent viscera, the contents of the stomach are suddenly precipitated into the general cavity of the abdomen, exciting general peritonitis. Ulcers situated on the anterior wall of the stomach are specially exposed to this danger, since in that situation adhesions can not easily be formed. The larger vessels of the stomach being deeply placed, escape the eroding action of the ulcer, unless the ulceration has proceeded deeply, nearly to the point of perforation. Furthermore, in the process of extension of the ulceration, the vessels resist longer, and become occluded, before yielding to the erosion. Now and then, arterial twigs are entered by a slough, or veins about the ulcer, which have become varicose, as is frequently the case, are destroyed by a superficial ulceration. Relapses are comparatively frequent. The cicatricial tissue, having low vitality, ulcerates from slight causes.

Changes, which have apparently some relation to the morbid process in the stomach, occur in other organs. It is clear, however, that certain diseases of the arterial system, as endocarditis, endarteritis, have an immediate connection, for embolism and thrombosis are important factors in the pathogeny of ulcer. In about one half of the cases, there is coincident pulmonary disease, very often tuberculosis. It is a popular notion that stomach-ulcers are transformed into cancer; it is true that cancer sometimes develops at the site of an old ulcer.

Symptoms.—There are three important symptoms of stomach-ulcer—pain, indigestion, and vomiting (hæmatemesis). It should be known that some very acute cases occur without symptoms. In apparently perfect health, an individual has a perforation of the walls of the stomach; an acute peritonitis is immediately lighted up; intense pain, vomiting of blood, and profound prostration occur, and death takes place in a few hours or in a day or two. The author has met with such a case. More usually ulcer of the stomach is a chronic malady and characterized by the existence for many months or years of the three symptoms mentioned. Although the pain varies in intensity and differs much in different cases, yet, on the whole, there is remarkable correspondence. In the largest number of cases the pain is felt in front, in or just below the xiphoid appendix; or in the left hypochondrium in the intercostal space between the sixth and seventh rib, occasionally; more frequently above the umbilicus, in the neighborhood of the pylorus. Posteriorly, and this position is even more important, the pain is felt in the region of the last dorsal or first lumbar vertebra, or under the angle of the scapula. The pain in front and behind seems to be continuous, as if it passed directly through the body. This is its distinctive character—a fixed, gnawing, burning pain, boring through from front to back, and occupying a space which the finger may cover. More or less pain radiates from this central and fixed pain, and is felt in the chest behind the sternum, in the intercostal nerves, in the cervico-brachial plexus, etc. Very great tenderness is experienced on pressure over the vertebra behind and the seat of pain in front. Corsets or a tight dress can not be borne, and, in sitting, the patient seeks a position more or less bent, to avoid the pressure of internal organs against the sore spot.

Besides these, already described, the patient suffers with attacks of gastralgia, sometimes of extreme violence, but they do not occur with any regularity. When the gastralgia comes on, the fixed pain is increased in severity, and pain of extraordinary violence radiates through the abdomen and chest. During these paroxysms, the action of the heart becomes very feeble, and the vital forces much depressed. An alarming syncope, or general convulsions, may ensue if the patient possess a highly sensitive reflex organization. As the attacks are usually due to the presence of indigestible food, they cease when the stomach is empty; but they also arise from cold, fatigue, mental and moral emotion—to the causes, indeed, of neuralgia elsewhere. The pain of stomach-ulcer—the fixed pain—is increased by taking food. In a majority of cases the increase of pain is experienced as soon as food enters the stomach; in a smaller proportion the exacerbation occurs in from fifteen minutes to a half hour; in others, the most severe suffering takes place when food is supposed to be passing through the pylorus, in about three hours after eating. The character of the food influences the production of pain—indigestible, especially irritating, articles causing greater suffering than bland articles. The increase of pain persists until the food is rejected by vomiting or passes the pyloric orifice. The pain caused by the presence of food in the stomach should not be confounded with the attacks of gastralgia, which may arise from hygienic and moral causes as well as improper food. Some cases of stomach-ulcer are free from distress of any kind; in fact, they continue for months and years with no more local disturbance than is produced by chronic gastric catarrh; but these must be regarded as exceptional. Vomiting is a frequent but not an invariable symptom; in a few instances it never occurs; in others it comes on late in the course of the disease. The vomiting is preceded and accompanied by pain, but, when the stomach is emptied, the pain ceases. Occasionally attacks of vomiting and pain occur when the stomach is empty; some glairy mucus, with or without blood, only, coming up with a good deal of straining, showing that the disturbance of the stomach is not due merely to the presence of food. If the vomiting persist, and there be much retching, some bilious matter may finally be brought up. But the great factor is unquestionably food, and especially undigested food; but more or less gastric catarrh is a constant element in cases of ulcer of the stomach. The time when the vomiting occurs may indicate the position of the ulcer. If the ingestion of food is followed immediately by pain, the ulcer is probably in the vicinage of the cardia. If situated in the greater curvature, there may be but little vomiting, and that will take place in about an hour after food; when near the pylorus, vomiting is an invariable symptom, and the pain is great, but the pain and vomiting do not come on until two or three hours. It must be admitted that these statements as to the time of the vomiting and the position of the ulcer are only approximately correct. Vomiting of blood is the most characteristic single symptom, but is not pathognomonic. It is absent in about one third of the cases. Hæmatemesis may occur only at the monthly period as a vicarious discharge, or merely as an accompaniment of the regular flow. Pain coming on after eating, vomiting of food mixed with blood, and then of blood only, is an extremely significant combination of symptoms. The vomited matter may consist only of blood, red or brownish red, when it comes up immediately; if retained for a short time, it appears in clots more or less blackish if acted on by the stomach-juices. When held in the stomach for some time, and the amount is small, it may present the well-known "coffee-ground" appearance; but if the quantity is large, and has been acted on by the gastric juice, and churned up by the movements of the stomach, it will then have a brownish-black, uniformly granular, and homogeneous aspect. As the vomiting usually occurs quickly after the blood is poured out, the ordinary and characteristic appearance is that of reddish blood partly coagulated. Coffee-grounds, blackish and brownish-black masses or particles, belong rather to cancer. The nutrition may or may not be impaired in gastric ulcer. The small perforating ulcer is often met with in young girls of rather full habit but lymphatic in type. The chronic ulcer of long standing, if small, may not affect the digestion sufficiently to lower the body-weight; but, if large, the digestion-space is so much abridged, that there must be a constant waste, which the primary assimilation is unable to supply. Much depends on the amount of loss by vomiting, and this is influenced somewhat by the inherent irritability of the stomach. The frequent recurrence of hæmorrhage also seriously impairs the nutrition and induces a cachectic state and a peculiar tint of the skin, which may be confounded with the earthy hue of carcinoma. The tongue may be clean, somewhat furred, red at the tip and at the edges, fissured, but there is no characteristic appearance. As a rule, there is obstinate constipation. Amenorrhœa is a frequent complication, due partly to the vicarious hæmatemesis and partly to the profound anæmia to which some patients are reduced.

Course and Duration.—The behavior of the acute and perforating ulcer has been sufficiently discussed. The chronic and common form has a very variable duration. Well-authenticated cases have existed ten years—an example of which the author has had under observation. From three to five years is a comparatively common period of duration. The chief reasons for their long-continued existence are, their essentially chronic character and the frequent changes in their condition—now increasing, now improving, almost cicatrized, then a change in the constitutional state of the patient, or indiscretion in food will re-excite ulceration in tissue almost or entirely repaired. At various periods in the course of the chronic ulcer there may occur a chill followed by fever, exquisite tenderness of the epigastric and umbilical regions, nausea, vomiting, constipation, a quick, small pulse, etc., symptoms of a local and limiting peritonitis. Some cases of chronic ulcer run an entirely latent course ; that is, there are no more pronounced symptoms than those of dyspepsia.

Termination.—A large proportion terminate in recovery—complete cicatrization, without any subsequent impairment of the functions of the stomach. The cure may be partial ; there may be adhesions contracted to adjacent organs, which alter the shape and impair the motions of the stomach ; contraction of the pyloric orifice, leading to dilatation and gradual inanition. The ulcer may cause death in various ways : there may be a gradual failure from pain, vomiting of food, vomiting of blood, and by the growth of lesions in other organs (cardiac disease, tuberculosis, etc.). Death may occur by hæmorrhage—according to Brinton five in one hundred so terminate. A considerable proportion—13·4 per cent.—die by perforation and consequent peritonitis. This unfortunate accident is announced by a sudden and great depression in the powers of life, and death by shock, or the prompt development of fatal peritonitis.

Diagnosis.—Notwithstanding a diagnosis may be made with great certainty in cases presenting typical symptoms, it may be very difficult in other cases. The maladies presenting similar symptoms are, ulcer and chronic gastric catarrh, gastralgia, hepatic colic, cancer, and chlorosis. In chronic gastric catarrh the pain after food is much less, and, in fact, in very many cases the distress is alleviated by taking food ; vomiting is occasional, and there is no vomiting of blood. The paroxysms of gastralgia may be the same as in ulcer, but the behavior of the two diseases, otherwise, is very different. Gastralgia is in paroxysms entirely, and between them the patient suffers but little, and does not always have pain after eating, vomiting, and relief by the rejection of food and the vomiting of blood. In hepatic colic the pain radiates from the region of the gall-bladder, suddenly terminates when the calculus reaches the intestine, and is followed by jaundice. During the attack, owing to the congestion of the portal system, there may be vomiting of blood, but it is never great in amount, and all the symptoms subside in a few days, the patient being free from any disturbance of the stomach afterward. In cancer, the age of the subject, the emaciation and cachexia, the tumor and enlarged lymphatic glands, the vomiting of coffee-ground and blackish and brownish-black material, instead of the red or brownish-red blood in large amount in ulcer, are the most characteristic differences. It is more difficult to separate chlorosis with amenorrhœa from ulcer of the stomach, because these subjects have the distress after food, the vomiting, and vicarious menstruation by the stomach. Under these circumstances of inevitable doubt, it were better to decide by therapeutic means. The case may be treated as one of gastric ulcer by an absolute low diet; if it is a case of ulcer, it will improve under this method; if a case of chlorosis, it will get worse—then a resort to iron and mineral acids will bring about a decided change for the better.

Prognosis.—Although the cure of ulcer may be confidently expected in favorable cases, yet such are the dangers from perforation and hæmorrhage that the prognosis must be regarded as serious. When tuberculosis and endocardial lesions exist, the gravity of the case is correspondingly increased.

Treatment.—The first and most important consideration is to give the stomach rest, which is accomplished by reducing the food taken to the minimum. An exclusive milk-diet accomplishes this object, while at the same time it contains the necessary alimentary principles for the support of the body. All rough, harsh, and coarse ingesta, such as oatmeal, brown-bread, and fruits, irritate the surface of the ulcer, and increase the existing ulceration, and retard healing. Starchy and saccharine foods are objectionable because they ferment, producing acid which is very irritating to the ulcerated surface. Milk should be given systematically—one gill (four ounces) every three hours, day and night, during waking, and it acts better if taken hot, 110° to 115° F. If it cause a sensation of heaviness or uneasiness, nausea or vomiting, the addition of lime-water will enable it to be better borne. The meat solution so strongly advocated by Leube, or that of Valentine, can be substituted for milk, if the latter prove repugnant to the patient or can not for any reason be used. To aid in supporting the powers of life, rectal alimentation may be employed. Foster proposes to relieve the stomach entirely for a time, supporting the powers of life by rectal alimentation, since the healing process is greatly promoted by giving the organ some days of absolute rest. The discovery of the utility of defibrinated blood, as a means of rectal alimentation, made by Dr. Smith, of New York, has added to our resources. The method consists simply in defibrinating the blood as soon as drawn at the shambles, and in injecting from three to six ounces morning and evening. If rectal alimentation is not employed exclusively, it should be combined with the milk regimen—for, the richer the condition of the blood, the more rapidly and perfectly can repair take place. As the destruction of the mucous membrane was originally brought about by the solvent action of the gastric juice, and as the irritation caused by this is the chief obstacle to healing, it is important to diminish the acidity and to keep the surface of the ulcer clean. These purposes are now accomplished by mechanical means, by irrigation of the cavity of the stomach by the siphon or the stomach-pump, as the same process is employed in other stomach-diseases; but caution is necessary in the use of the pump, lest the tube might cause a perforation. The same object may be accomplished by medicinal means—by the free use of the alkaline mineral waters. As regards the strictly medicinal remedies, the most important is arsenic in small doses, one to three drops of Fowler's solution three times a day. Next, named in the order of their relative importance, are oxide and nitrate of silver, in half-grain doses three times a day, and bismuth in fifteen-grain doses. If there be much pain, morphine in the hypodermatic mode; but, if the alimentation is proper, pain will hardly require attention. The regimen advised should be pursued for several weeks, or until such improvement is manifest as to indicate that cicatrization is pretty well advanced, when the diet may be very carefully enlarged by the addition of rice, soft-boiled eggs, animal broth, etc.; but the patient should be impressed with the importance of a simple dietary ever after.

Various new remedies have been used successfully in recent years. Resorcin is proposed for its local action, and exalgine for analgesic power—the former makes a superficial caustic action which prepares for healing, and the latter acts as a local anodyne, removing the irritability which is the chief cause of pain and nausea.

Two accidents in the course of stomach ulcer may require prompt action and the use of efficient means of treatment—hæmorrhage, perforation. In the former we have the application of ice to the epigastrium, large draughts of hot water, and sometimes of ice water in alternation. It is true that hot water is more effective in many instances; when so, it should be persevered in, and the cold stopped. To get the best results from hot water, there should be short intervals between the applications, so that the reactive contraction of the arterioles may take place.

For the relief of hæmorrhage certain systemic means are available. First, for immediate action at the point of injury: such astringents as tannic acid, and the various vegetable remedies containing it, are first in utility; but if the flow is strong and the state of the patient threatening, the more powerful astringents—tincture of the chloride of iron, solution of pernitrate, etc.—must be resorted to. The action of these iron salts on the blood coagula must not be overlooked, for when given in quantity, they form a hard mass sometimes difficult to dislodge, and hence increase the hæmorrhage.

To maintain a constant impression on the bleeding vessels, remedies that lessen their caliber, and at the same time slow the heart, are of decided benefit. Digitalis, aconite, ergot, and veratrum viride, especially the last, when the circulation is active, and barium chloride when there is a relaxed and atonic state of the vessels.

CARCINOMA OF THE STOMACH.

Etiology.—The points of election for the development of cancer in the intestinal canal, named in the order of their relative frequency, are the stomach, the rectum, the cæcum, the flexures of the colon. Of all the organs of the body, the stomach is most frequently the seat of cancer—more frequently than the uterus, which comes, strictly, next. As regards age, the majority of cases occur at fifty, but the disease may appear at any time from forty-five to sixty. It is very rare from thirty to forty. According to some authorities, cancer attacks the male sex by preference, but careful investigation shows that this view is erroneous, and that the two sexes are about equally affected. The well-to-do classes are said to be more liable to the disease than the poor, and the obese, hearty feeders, rather than the abstemious, but these are doubtful propositions.

Predisposition and heredity play an important part in the causation of cancer; they are, doubtless, the most influential factors. The inherited tendency may not be traced sometimes, when it exists, because of the behavior of the cancer-germ, skipping over one or more generations and appearing in subsequent ones. All other presumed moral and dietetic causes are rather fanciful.

Pathological Anatomy.—The forms of cancer occurring in the stomach are the following: scirrhus, or fibroid; medullary, or encephaloid; and the gelatinous, or colloid. As regards the site, the points of election are in sixty per cent. at the pylorus; in twenty per cent. at the lesser curvature; and in ten per cent. at the cardia. In the process of growth, extension is more apt to be vertical than transverse; but, when the growth is about the cardia or the pylorus, the new formation takes an annular direction, causing stenosis.

The initial changes in the development of cancer of the stomach are an increased vascularity and the presence of numerous white blood-corpuscles in the cylindrical epithelium of the gastric glands—as in ordinary inflammation—but the changes soon take a special direction and character. Rapid proliferation of the cells of the cylindrical epithelium occurs, and assumes a downward direction, penetrating the mucosa, the sub-mucosa, to the muscular layer, into which ultimately long, fibrous bands project. In the loose, submucous connective tissue the growth is most rapid, and here the nodules form in greatest numbers. The so-called cancer-cells—groups of proliferating cylindrical epithelial cells—lie imbedded in a fibrous stroma, made up from the connective tissue of the mucous membrane. Within and about the stroma an infiltration of small cells appears, and out of or within these are formed numerous minute vessels. Thus, in a short time from the beginning of the process, all of the anatomical elements of the mucous membrane are appropriated by the new formation. In the course of development of scirrhus, the connective-tissue element, the fibrous stroma, takes on a preponderating growth over the epithelium cells and the small cell infiltration, with its newly formed vessels.[*] It is in consequence of this preponderance of the connective-tissue element, whether in distinct nodules or in a dense annular mass, that it presents such a cartilaginous appearance on section. A large part of the stomach may be converted into a mass of scirrhus, of one or two inches in thickness, with nodules and protuberances of greater thickness projecting into the cavity. A dense mass, of half to an inch in thickness, much less nodular, may surround the pylorus or the cardia, leaving a considerable part of the mucous membrane of the stomach free from disease. No part of the mucosa exists after the cancer is developed; hence the internal surface of the stomach at that point is the surface of the cancer only, which is usually in an ulcerating state.

Medullary cancer, or encephaloid, differs from scirrhus in the less growth of the fibrous stroma, and in a much more luxuriant proliferation of the small cells and their associated vessels. Hence this form of the disease is softer, more vascular, and possessed of a greater power of rapid growth. Some parts of this form of cancer may, and usually do, retain the characteristic fibrous stroma of scirrhus. The internal or gastric surface usually consists of projecting nodules of softened cancer elements, which are easily detached and bleed readily. The ulceration which occurs in the exposed surface within the cavity of the stomach really consists in a process of fatty degeneration, the disintegration being produced by the solvent action of the gastric juice and the mechanical action of the food.

Colloid cancer differs from the other varieties in that a gelatiniform degeneration of the cancer cells takes place, giving the peculiar colloid appearance. The distention of the alveoli by this material dilates them so that they are larger than in other forms. This variety differs from the others also in that it is more widely diffused through the mucous membrane, and through neighboring organs, and is slower and longer in growth. It is also less common.

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[*] Waldeyer, Virchow's "Archiv.," vol. xli, p. 470, and vol. lv, p. 67, "Die Entwickelung der Carcinome." Also Förster, "Lehrbuch der path. Anat.," pp. 110–115, by Siebert, Jena, 1873. Rindfleisch, "Text-Book of Pathological Histology," Lindsay & Blakiston, 1872, p. 375, confirms Waldeyer's account of the origin of cancer in the mucosa. See also Rokitansky, and especially the great work of Cruveilhier, "Traité d'Anatomie Pathologique," where colloid will be found admirably delineated.

Cancer, like ulcer, by setting up local peritonitis leads to the formation of adhesions, which affect the shape, position, and motions of the stomach. Adhesions may fix the pylorus in or about its true position, but, when unattached, the weight of the cancerous mass may drag it down, even as low as the hypochondrium, and thus constitute a movable tumor. When the annular deposits form at the pylorus, a stenosis of the orifice and dilatation of the cavity are results. When the same formation occurs at the cardia, the stomach very much contracts, and the œsophagus immediately above dilates. In the vicinage of the connective-tissue bands, which stretch out through the subjacent elements, especially the muscular, considerable hypertrophy of these muscular elements at first results, but atrophy, from pressure of the newly formed connective tissue, finally occurs.* Those portions of the mucous membrane uninvaded by the cancer elements suffer chronic catarrh, in consequence, doubtless, of the continued hyperæmia. That from such a state of the mucous membrane cancer may develop, is a popular notion, not supported by any scientific data. It is true that hyperæmia of the cells of the cylindrical epithelium is apparently the starting-point of the development of cancer, but this hyperæmia is due to some peculiar irritation of the tissue. Cancer has developed from an old ulcer in some rare instances, but some remnant of gland-tissue must have remained.

Cancer of the stomach is usually primary, and in most of the cases is confined to that organ. It is rare, indeed, for the stomach to be secondarily affected ; but the author has seen a case in which cancer of the gall-bladder was followed by secondary deposits in the pylorus —an altogether unique case. In less than half the cases, cancer involves other organs as well as the stomach, and notably the liver, which is affected in about one fourth. Secondary deposits in the liver less often occur when the cardia is involved than when the lesser curvature and the pylorus are the sites of cancer.

The principal complications of cancer of the stomach are fatty heart, thromboses, pneumonia, tuberculosis, etc.

Symptoms.—In a few rare cases cancer has proceeded from its inception to its termination in the death of the patient without causing any distinctive symptoms. These are examples of cancerous infiltration of the mucous membrane in the greater curvature, the orifices being unaffected. In the first stage, before a tumor can be detected or the cachexia is evident, the symptoms present are those of a dyspepsia, which gradually assumes a more aggravated character. There is a good deal of pain from an early period, felt in the epigastrium usually, and increased by pressure, by food, and is also felt posteriorly. The pain is nearly constant, and, although at times more severe, there are not, as a rule, those violent paroxysmal attacks so often found in ulcer. The pain is acute, often burning, sometimes lancinating, but by no means invariably so; again, it is a sense of soreness and not severe pain; rarely it is entirely absent, according to Brinton, in eight per cent.*

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* Luton, "Cancer de l'Estomac," "Nouveau Dict. de Méd.," Paris, 1871.

The disorders of digestion increase with the duration of the disease; the appetite declines; distress after eating becomes greater; then attacks of acidity and pyrosis, with regurgitation of an acrid, acid liquid, come on. Emaciation and loss of weight proceed at a uniform ratio. If annular deposits have been occurring at the cardia, the patient early becomes conscious of a difficulty in getting food into the stomach, but he almost invariably refers the obstruction to a point higher up. As the case advances, the alimentary substances pass slowly down to the cardia, where they are arrested for a minute or more, some portions trickling through into the stomach, the rest slowly returned by regurgitation, with a distinct gurgling noise. Considerable pain is experienced—a burning pain usually—when the substances swallowed reach the cardia, and as they pass through it into the cavity. This passage through the narrowed orifice is, as a rule, distinctly recognized and accurately described. When the liquid or solid is disposed of, either by regurgitation or by entrance into the stomach, there is a feeling of relief, and the stomach digestion goes on with the ordinary facility. In cancer of the cardia, but a small portion of the mucous membrane is destroyed—the deposits being annular—and, as death takes place earlier by inanition than in any other form, there is not much interference with digestion, and these unfortunates suffer horribly from hunger. The epigastrium contracts and is drawn in toward the spine, owing partly to the exceeding general emaciation, and partly to the extreme contraction of the stomach.

The absence of hydrochloric acid from the gastric juice, as a symptom which must possess great value if correct, has been much discussed of late. It has been held that this sign is infallible; then, that there are so many exceptions as to destroy its value for diagnosis. The latest researches tend to show that the acid is absent in cases of cancer, but it is also absent in other states of the system. Wolff and Ewald † assert that the acid is absent from the gastric juice of old people. In a case of cancer, Hoonigmann and Noorden ‡ found the merest trace of acid. These facts show that this sign can not yet be relied on. Some of the difficulties attending the solution of the question are the chemical methods, for they are not only uncertain in themselves, but require high skill in application. The test lately proposed by Günzberg, one of the simplest, is as follows: Dissolve 2 grammes (30 grs.) of phoroglucine and vanilline, 1 gramme (15 grs.), in 30 grammes (1 oz.) of absolute alcohol. To a few drops of this solution add the same quantity of the filtered stomach-juice. The mixture should be evaporated in a porcelain dish over a spirit-lamp, but should not boil. So small a quantity of hydrochloric acid as 1 in 10,000 will be detected by the formation of crystals of a red color, or if half as strong streaked with red.

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* "Medico-Chirurgical Review," vol. xx, p. 479. Also Brinton, "Diseases of the Stomach."

† "Centralblatt. f. d. Therap.," 1888.

‡ "Zeit. f. klin. Med.," 1888.

It is said that in cases of cancer there is not enough acid present to turn methyl-violet blue. This reaction necessarily has a limited scope, and is not final as to the character of the acid.

The subject must be regarded as yet *sub judice,* although the facts warrant the statement that in cases of cancer of the stomach in persons of middle age the absence of hydrochloric acid may be regarded as a diagnostic sign of real value, but not conclusive.

In the other forms of cancer, instead of arrest at the cardia, the patient feels no distress until the alimentary materials reach the stomach, when nausea and other distresses begin. Vomiting is one of the most constant symptoms, occurring in three fourths of the cases. At first the patient brings up in the morning, with a good deal of straining, some tough, glairy mucus, and, it may be, a little bilious matter. Presently the vomiting comes on after eating; if the cancer is situated just below the cardiac orifice, and does not constrict it, pain, nausea, and vomiting, begin almost immediately after the food is swallowed. If the posterior wall is affected only, vomiting may not occur until late in the disease, and then may not be a very pronounced symptom. When the pylorus is affected, vomiting is a pretty nearly constant symptom, but it does not occur until some time after the food has reached the stomach—as a rule, not until two or three hours have elapsed. The vomited matters consist at first of the food in various stages of solution, then of mucus, containing sarcina and other minute organisms, and when the case is pretty well advanced there appear small brownish or brownish-black or chocolate-colored masses, of small size usually, which consist of decomposed blood. Vomiting ultimately occurs without the presence of food: it is then the form of vomiting entitled *vomiting of irritation.* Hæmatemesis is a frequent but not a constant symptom, occurring in somewhat less than half the cases (forty-two in one hundred, according to Brinton). If, however, the vomited matters were carefully searched for altered blood, it would probably be found present in nearly all cases. If the spectroscope were employed to examine all suspicious-looking particles, the absorption-bands between C and D, characteristic of hæmatin, would be often seen. Vomiting of blood in large quantity, as occurs in ulcer, is quite exceptional in cancer. Usually the blood is derived from small capillaries, but now and then sloughing takes place, and a vessel of considerable size is opened. The author has observed in some cases an enormous quantity of chocolate-colored, homoge neous, granular material, discharged both by vomit and by stool, in cases of cancer at the pylorus. The condition of the bowel is that of torpor, but toward the end ichorous matter passing down the intestine excites diarrhœa.

In one third of the cases observed by the author, salivation (not mercurial) was a symptom, and was either constantly or periodically present. The saliva had the ordinary appearance. The tongue is red at the tip and pointed, and is usually glazed.

The cachexia induced by cancer is characteristic. With the progress of emaciation, decline of strength is to be expected, but the subjects of the cancerous cachexia have an extraordinary sense of fatigue, which is felt when no exertion is made. The action of the heart is feeble, the pulse small, weak, and quick; the respiration somewhat hurried. The least exertion increases the number of the heart-beats and the respiration movements. The skin is thin, dry, harsh, and inelastic. The complexion is pallid, earthy, and bronzed, combined—a fawn color—and is strongly suggestive of the malady. Toward the end, œdema of the ankles appears—a mechanical result of the thromboses. The cachexia, though it may be late, never fails to come on.

A tumor is found in the proportion of eighty to one hundred cases. In some situations the tumors can not be felt, as when at the cardia, or in the lesser curvature, for here they are covered in by the left lobe of the liver. In other situations they may usually be detected by palpation—suitable attention being given to all the sources of error. The variety of cancer does not necessarily affect the question of a tumor; but a colloid growth may be diffused through the walls of the stomach, giving to the sense of touch the impression of thickening, and not of a defined tumor. On palpation, the tumor, if it exist, is felt to be hard, somewhat irregular, and nodular, if scirrhus, but softer and more elastic, yet well defined, if encephaloid or colloid. Even when in a position to be felt, it may elude search by reason of distention of the stomach, or of adhesions which may change the shape and position of the organ, or the presence of fluid in the peritoneal cavity—a result of the pressure of secondary deposits in the liver. Tumor of the liver, of the pancreas, movable kidney, aneurism, may be confounded with tumor of the stomach, and must be kept in view when making a diagnosis by exclusion. The relation of the tumor to the movements of the diaphragm should be noted; for a tumor of the stomach does not descend when the lung is inflated with air. When the pylorus remains free the weight of the neoplasm causes it to fall down, sometimes as low as the hypochondrium, and it continues movable. Tumors of the liver and spleen descend on full inspiration, but the pyloric tumor when adherent retains its position, and when movable is not influenced by the respiratory movements. When a scirrhus lies upon the aorta, a pulsation is communicated to it, but it is not an expansile pulsation, and there are none of the other signs of aneurism, yet mistakes of diagnosis are not infrequent.

Like ulcer, cancer may result in perforation and general peritonitis; in the formation of fistulous communications with the walls of the abdomen, externally; with the transverse colon, when there will be stercoraceous vomiting; with the thoracic cavity; but these are comparatively rare complications. Occasionally a large vessel is laid open, and death ensues from sudden and large hæmorrhage. In accordance with its nature, cancer tends to spread to contiguous parts by reason of immediate vascular communication. The cancer elements are much more frequently deposited in the liver than in any other organ. Ascites, icterus, thrombosis of the portal vein, etc., are the most important results of the implication of the liver. Extension of the disease also occurs by the lymphatics, and large nodular masses of degenerating mesenteric glands may be felt through the thin parietes of the abdomen during the life of the patient. The cervical lymphatics, just above the clavicle, also sometimes enlarge and afford valuable indications of the nature of the malady, even early in the course of the disease.

Tuberculosis of the lungs is a frequent complication of cancer of the stomach.

Course and Duration.—Cancer of the stomach is an essentially chronic disease. The average duration, according to Brinton, is one year; but the cases differ in duration according to the anatomical site. Named in the order of their fatality they stand as follows: cancer of the cardia, of the pylorus, of the lesser curvature, of the greater curvature. The maximum duration is three years.

Diagnosis.—The differentiation is to be made between chronic gastric catarrh, chronic ulcer, and carcinoma. In the early stages of ulcer and cancer it may be impossible to separate them from chronic gastric catarrh; but as these cases progress the points of difference become distinct. The following considerations will enable a correct differentiation to be arrived at: chronic gastritis may occur at any age; there is rarely any severe pain, and it is diffused over the whole organ; vomiting is only occasional, and then of alimentary matters, as a rule; there is no important variation in the body-weight, and no progressive emaciation. In ulcer the pain is severe, localized to a small point in front and behind; there is much vomiting and hæmatemesis, the blood coming up in considerable quantity, little or not at all altered. The subject of cancer is well advanced in life (from forty to sixty); the pain has a lancinating character, and is felt in one place, which is the same for each case, but differs in different cases; there is vomiting, especially vomiting of chocolate or coffee-ground masses of decomposed blood; above all, the presence of a tumor. The presence of an excess of *indican* in the urine has been held to be diagnostic, but, as Jaffé* has shown—and this observation has been confirmed by Hoppe-Seyler—the quantity of this substance is much increased in carcinoma of the liver, in obstruction of the intestines, and other diseases.

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* "Centralblatt f. d. med. Wissenschaften," p. 2, 1872.

Treatment.—Although cancer of the stomach is incurable, much may be done by treatment to render the patient's decline tolerable. The first and most important point is to regulate the diet. By the withdrawal of solid food, and the substitution of milk alone, or milk and beef-juice, the greatest relief is afforded, and for a time there may be a gain in weight, but of course this is not long maintained. If the diet is restricted to the articles mentioned, it should be supplemented by that important means of rectal alimentation, the injection of defibrinated blood. The burning pain is much diminished by washing out the stomach once a day with the stomach-pump, especially in dilatation from stenosis of the pylorus. By removing acrid and acid matters in this way, much straining efforts at vomiting will be saved.

Of all the remedial measures proposed there is no prescription which is so generally useful in these cases as equal parts of pure carbolic acid and tincture of iodine, of which one or two drops may be administered in water three times a day. For the vomiting only, a solution in cherry-laurel water of carbolic acid, or a combination of carbolic acid with bismuth in an emulsion, will be found effective. Nitro-glycerin, benzine, and bisulphide of carbon have been used, with advantage, to allay nausea and vomiting. The most effective means to relieve pain is the hypodermatic injection of morphine. The stomachal administration of the same agent is inefficient, owing to the diminished absorption power of the organ. Laudanum by enema, morphine in the form of suppository, or the endermic use of morphine, are preferable to the stomach administration. Great care is necessary in the prescription of anodynes, for the need grows rapidly, and the consumption becomes enormous, reducing the patient to a mental and moral weakness dreadful to contemplate.

Arsenic, in the form of Fowler's solution, one or two drops, three times a day, has considerable power to allay pain, and is not without influence in retarding the growth of epithelial cancer. As respects the power to relieve pain, the physiological basis for its employment is the action of arsenic, in toxic doses, on the nervous system of animal life. It has been repeatedly observed that sometimes, in large doses, no vomiting was produced, but coma and insensibility followed. A great many facts have now been accumulated, proving that cancer of epithelial origin may be greatly retarded in its growth by the persistent use of moderate doses—two drops of Fowler's solution *ter in die.*

The author's considerable experience in the treatment of carcinoma of the stomach warrants the statement that the best results are obtained by the persistent use of carbolic acid and iodine, in the form advised above, and of arsenic, in the form of Fowler's solution. It may not be needless to observe that these agents should not be given in one prescription—the carbolic acid and iodine together, the Fowler's solution at another time.

HÆMATEMESIS—HÆMORRHAGE OF THE STOMACH—VOMITING OF BLOOD.

Definition.—Hæmatemesis and vomiting of blood do not adequately name the malady, for blood may be swallowed and then vomited. Hæmorrhage of the stomach is the correct term.

Causes.—Rupture of a stomach blood-vessel is the essential condition of stomachal hæmorrhage, notwithstanding, under some circumstances, diapedesis of the corpuscular elements does occur. Sufficient blood must escape to excite nausea and vomiting. During an inflammatory stasis, considerable blood may escape from ruptured capillaries, but usually hæmorrhage is due to the giving way of vessels of some size; diapedesis, certainly, is quite inadequate to bring about the escape of much blood. There may be disease of the tunics of the blood-vessels sufficient to cause them to give way on slight increase of the blood-pressure. Furthermore, long-continued abnormal pressure will induce slow changes, without invoking other causes to account for their yielding should the pressure suddenly become greater. In this way may we explain the occurrence of gastric hæmorrhage in cirrhosis, acute yellow atrophy of the liver, yellow fever. Certain lesions, acting mechanically on the portal vein, bring about the same results—for example, an aneurism of the hepatic artery, a large calculus, or tumors in the neighborhood of the portal vein. Any obstruction of the portal vein may be the cause of blocking by a thrombus of a vessel returning blood from a certain part of the mucous membrane—the effect of this being the production of one or a number of superficial ulcers. Severe and protracted hæmorrhage may proceed from such erosions. Still more remotely is the occurrence of gastric hæmorrhage, caused by increased pressure in the portal system due to obstructive troubles of the lungs and heart. The hæmorrhagic diathesis may manifest itself in hæmorrhage from the gastric mucous membrane. Arrest of an hæmorrhoidal discharge, which has continued for a long time, is supposed, by a sudden increase in the blood-pressure within the portal system, to be a cause of hæmorrhage of the stomach.

According to the statistics of Handfield Jones, in seventy-two cases of hæmatemesis there were fifty-three females to nineteen males—showing a great preponderance in the female sex. As regards age, from twenty to forty there were nine males and thirty-six females, and after forty, eight males and fourteen females. These facts indicate that vicarious menstruation through the stomach must be relatively frequent. As in forty the existence of ulcers seemed probable, it is rendered pretty certain, by these figures, that ulcer is the most common cause of stomach hæmorrhage.*

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* "Medico-Chirurgical Transactions," vol. xliii, p. 353.

Pathological Anatomy.—More or less coagulated blood, acted on by the acids of the gastric juice to a varying extent, is found in the stomach. It is often impossible to discover the source of the hæmorrhage, unless the hæmorrhagic erosions, already alluded to, have formed. They are usually situated in the neighborhood of the pylorus. When a large vessel has given way, the rent can usually be found with a coagulum in it.

Symptoms.—When a hæmorrhage occurs sufficient in amount to produce definite symptoms, the patient experiences a sensation of warmth in the stomach, while the periphery is cool or cold ; distention, nausea, faintness. If the hæmorrhage is large, coming suddenly from a vessel of considerable size, without any apparent cause, the patient turns sick, faint, pallid, and cold, the stomach is distended, and then vomiting sets in, the blood rushing up in a full stream through the mouth and nose, or if less in amount it comes up by successive acts of vomiting. The faintness usually increases at the sight of blood, and only passes off on the cessation of the bleeding. In rare instances a large hæmorrhage occurs, the stomach is fully distended and returns a perfectly flat percussion-note, the patient becomes pale and cold and faint, or he actually does faint and is convulsed, without any vomiting, the blood subsequently passing off by stool. A patient enfeebled by disease may be suddenly carried off by a hæmorrhage in the stomach without vomiting. It not unfrequently happens that, when the blood comes up with a sudden gush, some is carried into the larynx, where it excites coughing, and hence may appear to be coughed up. This fact leads to erroneous interpretation of the nature of the case, and confusion as to the source of the hæmorrhage. The appearance of the blood is different according to the time it has been acted on by the gastric juice. If it comes up at once in large quantities, it is partly fluid and partly coagulated, like ordinary blood ; but, if it has been retained, it has a blackish, or brownish-black, or chocolate appearance, and is then rather granular in structure. If but little blood has escaped and slowly, it presents the "coffee-ground" appearance. The gastric juice decomposes the hæmoglobin and sets free the hæmatin, which gives the color to the vomited matters. In concealed hæmorrhage of the stomach, the blood passing into the intestines, and in intestinal hæmorrhage, the same phenomena ensue : there occur sudden distention of the abdomen and colic-like pains, faintness or actual fainting with its attendant symptoms, if the loss of blood be large, and the stools of tarry-like material, altered blood, at first mixed with ordinary fæces, and then consisting of the decomposed blood only. As narrated in the previous article, the author has observed chocolate-colored material in large amount discharged by stool. It assumes this appearance when acted on by alkaline fluids, after the effect of acids. If this be correct, we have a means of determining whether any given discharge of blood originated in the stomach or intestine. Blood so colored may be vomited, but it comes up after the stomach is emptied, and is forced by the act of vomiting from the duodenum. A very singular result of stomach hæmorrhage is amaurosis, first observed by Graefe, then Fikentscher, and afterward by Hutchinson. No explanation that has been offered satisfactorily explains the occurrence of double, incurable amaurosis after hæmorrhage from the stomach.

Course, Duration, and Termination.—Occasionally vomiting of blood is fatal, as when an aneurism ruptures into the stomach. Although the patient may be faint, cold, and convulsed, yet hæmorrhage of the stomach is rarely fatal, and the patient slowly emerges from the condition of anæmia. The pain of ulcer and cancer is often much relieved by vomiting blood; but the case of ulcer may be made much more serious by it in all other respects. Hæmorrhage due to cirrhosis of the liver far advanced may be difficult or impossible to control, and may add materially to the dangers of the case, or may cause death by exhaustion.

Diagnosis.—The juices of colored fruits (of black raspberries, for example) may be mistaken for blood, especially when vomited in the night. The author has encountered several cases of this kind. The microscope or the spectroscope may be invoked to decide. Much greater difficulty must exist in determining the source of the blood, whether swallowed and vomited, or derived from the stomach or lungs. An examination of the nares will usually demonstrate the origin of the bleeding, if the blood proceeds from any part of the nasal mucous membrane.

Blood from the lungs has an alkaline reaction, is aërated, a bright red, and may contain mucus or pus. Blood from the stomach is acid in reaction; when acted on by the gastric juice, is blackish, brownish-black, or chocolate color, and is not aërated, and may be mixed with food. The act of vomiting brings up the blood from the stomach, of coughing from the lungs (coughing may attend vomiting of blood, and vomiting—the patient swallowing blood coming from the lungs—may attend pulmonary hæmorrhage). The previous history of pulmonary disease and the existence of moist *râles* at the time of the hæmorrhage indicate the lungs to be the seat of the hæmorrhage, and the absence of all the physical evidences of fullness of the stomach negatives the idea of stomachal hæmorrhage. The attack begins in the lungs, by a sense of heat under the sternum, by a soreness in some locality, and by a sense of constriction of the chest; in the stomach, by a sense of fullness and actual distention of the stomach, followed by nausea. After the attack of pulmonary hæmorrhage the patient experiences soreness at the seat of the hæmorrhage; there is more or less elevation of temperature, often a pneumonia or bronchitis of small extent; moist *râles*, and the expectoration for several days of small, brownish-bloody sputa. After the hæmatemesis, only the depression and anæmia are present except stools of altered blood, which are usual.

Treatment.—The hæmorrhage, which is a vicarious menstruation, is relieved by diverting the flux to the uterus, its natural outlet. This is best accomplished by the use of the appropriate emmenagogues during the interval, of hot sitz-baths and hot vaginal douches, at the time of the expected flow. In the case of married women, leeches may be applied to the cervix uteri at the time of the menstrual molimen. When due to arrested hæmorrhoidal discharge, leeches should be applied to the anus, and aloes be administered.

When an impoverished condition of the blood exists, or when the so-called hæmorrhagic diathesis is the cause of hæmorrhage, effort must be directed to improve the composition of the blood, and to elevate the tonus of the vessels. When the hæmorrhage is occurring, the most absolute repose must be enjoined ; the patient should swallow as rapidly as possible pellets of ice ; ergotin should be injected subcutaneously, as much as three to six grains at a time, and it may be repeated as often as necessary ; a bag of ice should be put on the epigastrium ; and large draughts of iced alum-whey should be swallowed every few minutes. Ligatures around the thighs, tied tightly enough merely to stop a part of the venous blood in the lower limbs, is an excellent adjunct to the measures above proposed. If this is not done, the legs should hang down out of the bed, and the shoulders should be somewhat raised. The salts of iron (chloride, nitrate, subsulphate) may be administered for their styptic effect. A teaspoonful of the tincture of the chloride can be given in four ounces of ice-water. An objection to these ferruginous styptics is the very voluminous and nauseating coagula which they form, and which are apt to excite vomiting. Brandy is an excellent local astringent, and is generally serviceable in these cases, owing to the syncope. The stimulant is beneficial in raising the arterial tension, by furnishing a force for the vaso-motor system, which is in a state of paralysis. Tannic acid is a safe styptic, which can be used frequently and in relatively large (ten grains) quantity. Sulphuric acid may be employed successfully, and this has the advantage that a small quantity imparts astringent property to a large amount of water. Next to alum-whey it is the most efficient hæmostatic. If vomiting is obstinate, the one sixteenth grain of morphia hypodermatically will stop it, and contribute materially to the arrest of the hæmorrhage.

If the hæmorrhage has been sufficient to cause dangerous syncope, inhalation of nitrite of amyl may arouse the failing heart, or the injection of digitaline may be tried. Leube advises the subcutaneous injection of ether—a syringeful every few minutes—in cases of dangerous syncope from the hæmorrhage. Very great care is subsequently required in the alimentation, and in the use of remedies to remove the anæmia. Only milk should be permitted for some days; but this may be supplemented most advantageously by the rectal injection of defibrinated blood.

DILATATION OF THE STOMACH.

Causes.—Dilatation of the stomach is most frequently produced by stenosis of the pylorus. The great cause of narrowing of the pyloric orifice is cancer, but it may be due to chronic inflammation, hyperplasia, and subsequent contraction of the submucous connective tissue, or to hypertrophy and contraction of the muscular elements—the so-called sphincter—of the pylorus. These forms of local disease, limited to this locality, are excessively rare, while cancer is common. Exterior pressure, as of cancer of the pancreas, a floating kidney or other tumor, may cause stenosis of the pylorus and subsequent dilatation of the stomach. Dilatation of the stomach may be the result of excessive indulgence in the use of fluids, notably of beer. The author has observed several cases, in beer-drinkers, who drank ten, twenty, even forty, glasses of beer habitually every day.

Pathological Anatomy.—When stenosis exists at the pylorus, the whole organ is dilated, often enormously so, but the enlargement is not universal and uniform from the beginning; the dilatation commences in the fundus. With the development of the stenosis there ensues hypertrophy of the muscular layer, in accordance with the well-known pathological law. In dilatation without stenosis of the pylorus the muscular layer is thinner than normal, pale in color, and more or less advanced in fatty degeneration; the mucous membrane is, also, thin, pale, and without rugæ. Stenosis of the pylorus is caused chiefly by cancer, and hence the lesions peculiar to this new formation will be present. If ulcers have been excavated at the margin of the orifice, have subsequently coalesced, and cicatrized, the results of the contraction of the cicatricial tissue will be seen in a distorted and contracted pylorus.

Symptoms.—When stenosis of the pylorus and dilatation of the stomach are results of cancer formation, the symptoms of dilatation are quite dominated by those of cancer. It is necessary, here, to discuss the former only. The symptoms are those of chronic gastric catarrh, or of dyspepsia. There are three signs in addition to those of dyspepsia, which indicate dilatation of the stomach: rather persistent vomiting; return of food partly chymified and partly undergoing fermentative and putrefactive changes; the physical evidence of enlargement. The cavity having greatly increased capacity, enormous accumulations may take place, and hence when vomiting occurs the amount discharged will be great. The attacks of vomiting are more frequent than is usual in ordinary cases of dyspepsia, and they may become habitual. Regurgitation is a common symptom—particles of partly digested aliment, acid, acrid, and offensive, and foul gases, compounds of hydrogen with sulphur and phosphorus, coming up. In these acid and pasty materials is found the parasite *Sarcina ventriculi.* It is not yet known whether this minute organism is a cause or a consequence of the conditions present; but it is so often associated with dilatation of the stomach as to have some diagnostic value. The bowels are torpid, the fæces dry. The nutrition is much impaired in consequence of the insufficient conversion of the food, and the diminished absorption. Hence the patients affected with this malady waste, and, as the blood is deficient in water, they suffer from muscular cramp, chiefly of the flexors. These cramps were first described by Kussmaul (Leube), but the author has repeatedly observed them in cancer of the stomach, in diabetes, etc., and everybody knows that they occur in Asiatic cholera, the same cause, dehydration of the blood, operating in all these maladies to produce them.

The physical signs of dilated stomach are as follows: On inspection, an abnormal fullness and prominence of the whole stomach region will be seen; on percussion, the signs vary according to the state of the organ; if empty, a tympanitic percussion-note, of a somewhat metallic quality and extending from the sixth intercostal space to or below the umbilicus, is developed; if full, it is high pitched and flat, and, on assuming the upright posture, there is a zone of dullness at the lower part of the space which in the recumbent posture returned a tympanitic note. On auscultation of the dilated stomach, there is almost always heard a good deal of *succussion*—splashing of the fluid in the cavity—when the body is suddenly and strongly shaken. Placing the stethoscope over the pylorus, and smartly compressing the left hypochondriac and lumbar regions, the splashing of the contents of the stomach can be readily heard. The dimensions of the stomach when distended may be demonstrated by causing an abundant evolution of carbonic-acid gas from the reaction of sodium bicarbonate and tartaric acid—a solution of the former being given and followed by a solution of the latter. Another means of diagnosis consists in passing the stomach-tube, and noting the point at which it may be felt through the abdominal parietes.

Treatment.—The first and most important duty is a careful adaptation of the diet to the conditions present. The form of alimentation suitable to these cases is "dry diet,"* a diet without fluids. The quantity of other foods should be small, and as far as possible "water-free."

As paresis of the muscular layer of the stomach is an important factor in the dilatation, means must be employed to correct this. Strychnine hypodermatically, in the epigastrium, is an excellent expedient. Tincture of nux vomica and tincture of physostigma are effective remedies—ten to twenty drops of each—three times a day before meals. Great benefit is obtained from the use of galvanism, one electrode placed just beneath the mastoid process and the other at the epigastrium, and a mild current (from five to twenty cells of Siemens and Halske), slowly interrupted, passed through the pneumogastric. Fermentation should be prevented by the use of the sulphites, carbolic acid, etc., but especially by abstaining from starchy and saccharine substances, which produce a great quantity of carbonic-acid gas. The decomposing foods, the fat acids set free by the fermenting butter and other fats, and the unhealthy mucus which is poured out in great quantity, keep up irritation which renders futile the use of the ordinary remedies. This fermentative and decomposing mass must be removed from the stomach. The expedient first advocated and employed by Kussmaul—washing out the stomach with the pump or siphon—has proved to be useful, but it does not maintain the same position, as a therapeutical means, as on its first introduction. Recently Kuster * has opposed its use on several grounds, and advised the treatment by muriatic acid, Carlsbad salts, and nitrate of silver. If the stomach-pump or siphon be used, the stomach should be thoroughly washed out every day. The author can not doubt that, if an emetic is first given, and is followed by an active saline cathartic, the stomach will be thoroughly emptied, and as efficiently as if the stomach-pump were employed. Then, if distention be avoided, a suitable diet enjoined, and remedies to promote contraction of the muscular layer prescribed, the best results can be obtained of which our present resources will admit.

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* See my treatise on "Materia Medica and Therapeutics," article "Alimentation in Disease."

DISEASES OF THE INTESTINES.

CLINICAL EXAMINATION—INSPECTION—PALPATION.

In proceeding to examine intestinal disorders, a careful inspection of the abdomen in all its relations should be made. The patient should be placed on the back, hands folded under the occiput, and the lower limbs extended easily, without being tense. The muscles of the abdomen and back should, also, be relaxed, and the skin of the abdomen bared that every reflection of light from it can be noted. A good northern light should fall upon the parts, and the observer disconnecting his mind from all other subjects, should look carefully over the surface catching the reflection of light from every point. In this way, as the author fully believes, new formations, enlarged or-

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* "Allgemeine med. Central-Zeitung," 1876, No. 98.

gans, etc., may be seen in outline on the skin, photographed as it were, when the most practiced skill in palpation and percussion may fail to develop a lesion of any kind.

An examination of the stools is an imperative duty in all cases of diseases affecting the canal. This examination must not be merely perfunctory, but every change in color, consistence, in the products of digestion, and in odor, should be noted. In intestinal diseases, the formation of peptones, the solution of fats, and the reaction which in normal conditions occurs between the bile and the pancreatic juice, may be disordered or prevented, and therefore need to be understood.

The peculiar alkaloidal substances known as *ptomaines* and *leucomaines* which are formed in the intestines—the former from the products of decomposition of animal and vegetable substances and by the action of pathogenic microbes, and the latter by a process akin to a physiological one and yet no doubt due to the vital activity of micro-organisms. Ptomaines formed in the intestinal canal are now supposed to be causative of diseased processes, in severity varying from a mere transient sick headache to an essential fever, such as typhoid.

Palpation and percussion are of exceeding value in determining states of the abdominal organs, as to outline, texture, position, etc. Organs that are movable can be detected by *percussion* as absent from their normal place, and by *palpation*, present in some unusual position. Organs, movable, may be pulsating; but as a rule a pulsating tumor has a fixed position, as, for example, an aneurism of the mesenteric artery may be somewhat movable; whereas an aneurism of the aorta or cæliac axis is fixed. Auscultation has limited use in these affections, but may have a place in diagnosticating aneurism, tumors of unknown kind, and obstructions of orifices.

Besides the modes of study above indicated, there are methods partly surgical to which passing reference may be made—filling the large intestine with fluid to ascertain its freedom from obstruction, or with hydrogen gas to demonstrate a rupture of continuity, or with some chemical which by a reaction may show the position of some special lesion. These are mentioned that such resources be not overlooked in doubtful cases, or in emergencies.

CATARRH OF THE INTESTINES.

Definition.—Catarrh of the intestinal mucous membrane may exist in the *acute* or *chronic* form. It receives different designations as it affects the various divisions of the intestinal tract. Catarrh of the duodenum is *duodenitis*; of the ileum, *ileitis*; of the colon, *colitis*; and of the ileum and colon together, *ileo-colitis*. When it is limited to the cæcum it is called *typhlitis*, and when to the rectum, *proctitis*. Again, the designation is derived from some special characteristics, as *cholera morbus*, *cholera infantum*, etc.

To avoid repetition, those points in the morbid anatomy in which these several forms agree may be first described with advantage.

Pathological Anatomy of Catarrh of the Intestines.—In the catarrhal process, there ensues first hyperæmia of the mucous membrane, which is manifested by redness, swelling, and œdema; next, nutritive alterations, which consist of granulation of the protoplasm, overgrowth and desquamation of the epithelium. The injection occurs most decidedly about the glands, but it may be uniformly diffused, the whole surface affected, or the redness may be in patches and restricted to particular localities. One result of active hyperæmia is rupture of capillaries and extravasation of blood; another is increased secretion and exudation, consisting of the products of glands, abnormally active, desquamating epithelium, proliferating cells, and migrating white corpuscles. In these changes consists the morbid anatomy of an acute catarrh of a mucous membrane.

In *chronic catarrh*, which succeeds to the acute form, generally, the changes are similar, but possess also special character. Long-continued hyperæmia induces changes in the vessels—over-distended they remain enlarged, the veins tortuous and varicose; remains of old extravasations of blood are seen in a brownish, slate-colored pigment deposit, most abundant in the villi. The mucous membrane continues swollen and œdematous; the cells of the epithelial layer are altered in respect to their nuclei and protoplasm, which have become cloudy and are more or less advanced in fatty degeneration. The glands and agminated follicles become prominent from an excessive formation and accumulation of their contents; as a result of the pressure of proliferating cells, necrosis occurs, and sloughs separate, leaving ulcers; or the glands remain prominent and brownish and slate-colored from changes in previous extravasations. The mucous membrane is covered with a tenacious mucus rich in pus-cells, which strongly adheres, or with a more abundant and less tenacious purulent exudation. Owing to an accumulation of their contents, the agminated patches with solitary follicles are enlarged, their orifices appearing as minute black points, the whole forming a very characteristic appearance.

In chronic catarrh the anatomical alterations are not limited to the mucous membrane and its glandular appendages. The hyperæmia extends to the mucosa; its vessels, especially the veins, enlarge, and the connective tissue, in some situations, undergoes hyperplasia and thickens, forming prominences. Instead of hypertrophy, an atrophic change may result from chronic catarrh, but a very great duration of the disease and the immaturity of early life are necessary.

The muscular layer of the intestine, if a catarrh has long persisted, may undergo hypertrophy, and, in rare cases, to such an extent as to encroach on the cavity and greatly lessen the capacity of the bowel.

CHOLERA MORBUS.

Definition.—An acute catarrh of the stomach and intestines, of sudden onset, and manifested objectively by vomiting and purging. It is also called cholera nostras, sporadic cholera, etc.

Causes.—Climatic influences are the most important. It is a disease more especially of summer and early autumn, although it may occur under certain circumstances at any season. Tartar emetic, elaterium, and other irritants will bring on vomiting and purging not to be distinguished from a severe cholera morbus. Irritants of all kinds, unripe fruits and vegetables, fermentation of foods in the stomach, will excite an attack.

Pathological Anatomy.—Death may ensue without there being any defined alterations of structure. In ordinary cases there are present the changes of acute gastro-intestinal catarrh; the mucous membrane hyperæmic; the epithelium desquamating; the glands swollen and prominent; the blood thick and of a prune-juice color; the serous membranes everywhere dry, sticky, and coated with desquamated epithelium; the kidneys hyperæmic, the epithelium of the tubules also being cast off; the muscles of the body becoming granular, etc. —the morbid anatomy, indeed, of true cholera, except in degree.

Symptoms.—An attack of cholera morbus may be preceded by some diarrhœa, nausea, a coated tongue, and general *malaise* for a day or two, but usually it sets in suddenly and with violence. In the night, as a rule, and usually after midnight, the patient is awakened by a chill or a sense of chilliness, some intestinal pain (colic) and nausea, and vomiting then begins; or, without any premonition, the patient awakes with intense nausea, and then vomits immediately. The vomited matters at first consist of the ordinary contents of the stomach. Simultaneously, purging begins, the first evacuation containing more or less of ordinary fæces. Presently the matters discharged by vomit and stool are liquid, whitish, or of a green or yellowish tint, consisting of mucus and sero-mucus. In the severe cases, approximating to the true cholera type, the matters vomited or passed by stool are copious, thin, whitish, odorless, or having a faint mouse-like odor, and consist of blood-serum with mucus and cast-off epithelium (rice-water discharges). The discharges occur in quick succession, and so enormous is the loss of material that in an hour or two the patient may be so much reduced as to be unable to rise from the bed; the body shrinks, the face becomes pinched and cyanosed, the surface cold and covered with a clammy sweat; the hands shrivel and have a sodden appearance; the voice is husky, the tongue is cold, the breath is cold. The patient is tormented with an intolerable thirst, but the drink is rejected as soon as swallowed. The urinary secretion rapidly diminishes in amount, and in the worst cases is suppressed. The urine contains traces of albumen, casts of the tubules—the desquamated epithelium—and is deficient in the amount of urea and salts. The effect of this enormous waste from the intestinal canal is to diminish the water of the blood, and hence to relax the circulation. The action of the heart becomes so feeble that the pulse may not be felt at the wrist. Another result of the dehydration of the tissues is the occurrence of cramps, especially in the muscles of the calf, and they cause severe suffering, the patient crying out when they come on. They may occur in the muscles of the upper extremity, and also in the muscles of the neck. In some cases, enormous accumulation of the rice-water material may take place because of a paralytic state of the bowel, and no discharge occur by vomit or stool, yet the patient passes quickly into collapse.

From the simplest case of cholera morbus, which ends spontaneously when the stomach and intestines are emptied, up to the severe algid form, there are numerous intermediate examples of every degree of severity.

Course, Duration, and Termination.—The subsequent clinical history of the cases depends much on the severity of the attack. The mild case terminates without treatment, and the next day, although somewhat weak, the patient is about as usual. In the severer cases, after several hours the number of the evacuations lessens, and their character is changed, the skin becomes warm, the pulse rises, and the normal is presently restored, but the mucous membrane remains sensitive, and care in alimentation is necessary for several days. In the severest cases—those of the cholera type—recovery from the algid stage is gradual, reaction comes on slowly, but passes the norm into a fever, of type remittent and of character typhoid, which may continue a week or more. In the fatal cases, the mode of dying is by collapse, or in the secondary fever by exhaustion.

The cases are very uniform, but differ much in severity. The duration is from a few hours to two or more days, and, in the rare cases of secondary fever, to two weeks. The termination is in a great majority of cases in health, the mortality being about three per cent. of uncomplicated cases. An attack of cholera morbus may be the mere prelude to an acute diarrhœa or dysentery, more frequently the latter. An attack of cholera morbus may be the mode of dying from chronic interstitial nephritis.

Diagnosis.—The phenomena attendant on cholera morbus are so characteristic that a mistake of diagnosis would seem to be difficult. During the existence of a cholera epidemic, the severer cases of cholera morbus may be mistaken for cholera, but, as they do not differ in any respect, not even in morbid anatomy, there need be no attempt at differentiation. Cholera morbus, a substantive affection, may be confounded with choleriform attacks due to uræmia. The distinction is to be made by reference to the previous history, the presence of albumen and casts in the urine, and the cerebral symptoms, which, in some form, occur in uræmia.

Treatment.—In simple cholera morbus due to the ingestion of some irritating or indigestible food, or to fermenting materials, no treatment may be necessary. When the cause is removed the morbid action ceases. In the more severe cases prompt action is necessary, especially when cholera is prevalent. No remedy compares in efficiency to the hypodermatic injection of morphine and atropine—$\frac{1}{8}$ to $\frac{1}{4}$ of a grain of the former and $\frac{1}{120}$ of a grain of the latter.* Those entirely unaccustomed to the action of opium—women, and men of the nervous and impressionable type—should receive the smaller dose. In many cases, a single injection suffices to terminate the attack. The repetition of the injection will depend on the severity and persistence of the seizure, and on the susceptibility of the patient. It is usually better not to repeat the injection within the hour. The effect which it has is most striking: the vomiting and purging cease, the pulse rises, the surface becomes warm, and the cramps are no longer felt. It is rare, indeed, if these results are not obtained promptly, rendering unnecessary any subsequent treatment except some correcting medicine. In the cases of the cholera type, the patient passing into the algid stage, additional means may be necessary. The use of chloral hypodermatically with morphine is then remarkably beneficial. The author has observed that under these circumstances chloral will relieve the cramps and bring about reaction, when morphine, alone or with atropine, had seemed inadequate.

Other means of treatment may be employed in conjunction with the hypodermatic injections, or without them. Sinapisms of large size should be applied to the abdomen, but not allowed to remain longer than sufficient to produce a sensation of burning, or the appearance of redness. Pellets of ice may be repeatedly swallowed. Iced champagne, very dry, will sometimes be retained when other things are rejected. Carbonic-acid water and effervescing soda-powders are very grateful and also serviceable. The medicines most easily borne and most efficient are combinations of the mineral acids and opium, of which, the well-known Hope's mixture is a type. Diluted sulphuric or muriatic acids with the tincture of opium in camphor-water, are the best of these combinations. The mistake is frequently, indeed, usually made, of giving the mineral acids in too large doses, and hence they are immediately rejected. From two to five drops of diluted sulphuric, or the same dose of diluted muriatic acid, and the same quantity of tincture of opium, should be given from every half hour to every two hours, in a sufficient quantity of ice-water. An acid solution is much more grateful, and also more easily borne, than any other kind of medicine. Carbolic acid alone, or in a mixture with bismuth, is an efficient means for arresting vomiting. Besides its properties as an antiferment, it has a local anæsthetic action on the terminal filaments of the nerves in the mucous membrane. The effects of carbolic acid, creosote, and other agents of the same kind, are exerted on the stomach chiefly, and hence are less useful in affections of the intestines. Iodine tincture, and carbolic acid, in equal parts—a half grain of each—every half hour, is an effective combination, of great utility in irritable stomach. To these may be added such antiseptic remedies as naphthalin, resorcin, saccharin, etc., which have especial value in cases characterized by abnormal fermentations, decomposition of foods, and the pullulation of micro-organisms.

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* "Manual of Hypodermic Medication," fourth edition. Philadelphia: J. B. Lippincott & Co., 1882.

Very small doses of calomel—one twelfth to one sixth of a grain—have remarkable sedative effect on the gastro-intestinal mucous membrane, relieving vomiting and suspending the purging. It is often given with opium, with rhubarb, piperine, etc., but such combinations, except that with opium, are of doubtful utility. Aromatic and astringent remedies are much used in various combinations to arrest vomiting and purging. Tincture of rhubarb, tincture of calumba, and tincture of opium, make an effective remedy. One of the most generally useful and certain remedies for attacks of cholera morbus is chlorodyne. As a secret, proprietary remedy it should not be prescribed, but one of the more accurately prepared imitations of the original compound can be substituted. There can be little doubt now that this is a fortunate combination of remedies, adapted to the treatment of gastro-intestinal maladies having the choleriform character.

CHOLERA INFANTUM.

Definition.—An acute gastro-intestinal catarrh, occurring in children during the period of the first dentition, and characterized by vomiting, purging, and considerable febrile excitement. It is also called summer cholera and "summer complaint" in domestic practice.

Causes.—Early life—the first two years—owing to the various phases through which the organism is then passing, is the period for cholera infantum. Bad hygiene is the great factor—including damp, ill-ventilated, and confined houses, air contaminated by cesspool and sewer emanations, continuous high temperature, and improper food. Feeding infants the coarse food of adults, or confining them to a diet composed almost entirely of starch, are most fruitful causes of an outbreak of the disease, the other conditions being present. This peculiar form of gastro-intestinal catarrh occurs chiefly in cities, in low, malarious localities, and is especially frequent on this side of the Atlantic. But Berlin has the bad preëminence, according to Lombard,* of surpassing the American cities in "the frequency of the cholera of infants." Recent investigations tend to show that cholera infantum is caused by an infective organism—a specific microbe. It is in a high degree probable that these organisms or microbes generate a peculiar poison—a ptomaine—to the action of which the phenomena of the disease are due—probably *tyrotoxicon*, as shown by Vaughan.†

Pathological Anatomy.—The changes occurring in cholera infantum are those described under the general head of catarrh of the intestines. The implication of the solitary glands and the agminated (Peyer's) patches is somewhat more decided than is there stated, probably, but otherwise the description there given is accurate. A marked degree of cerebral anæmia is represented in a venous stasis, and a good deal of fluid in the subarachnoid spaces.

Symptoms.—This disease sets in by two modes of onset: with preliminary symptoms; suddenly. Usually there are prodromes, the child becoming restless, irritable, feverish, before any bowel symptoms are manifest, then diarrhœa comes on, vomiting occurs, and the disease is fully developed. In other cases diarrhœa has persisted several weeks with the usual symptoms, and gradually the phenomena of cholera infantum are added. Again, the disease is suddenly developed: the child, in full health, is attacked, without any preliminary symptoms, with the characteristic vomiting and purging. The first evacuations contain more or less fecal matter, but soon the characteristic watery stools make their appearance. These are so thin as to soak into the napkin, leaving a greenish or greenish-yellow stain, and having an odor of rotten wood, or indeed having but little odor. With these stools are particles of curd, or undigested food passed as swallowed, or yellowish masses of mucus turning green on exposure. Simultaneously vomiting occurs of any food or drink swallowed, and with these matters a quantity of sero-mucus, acid, neutral, or even alkaline, according to the time of the vomiting. Usually anything taken into the stomach —water or mother's milk—is rejected immediately; the retching continues, and the mucus coming up after the food is acid; further retching brings up some serous fluid, which is neutral, and alkaline if it comes from the duodenum. Prolonged retching brings up not only the contents of the duodenum, but mucus and bile from the gall-bladder. The loss by the gastro-intestinal mucous membrane induces rapid wasting. In a few hours the body shrinks remarkably, the eyes are sunken and half closed; the mouth remains half open, the lips dry and cracked, and bleeding, for the infant feebly picks at the fissures; the face is shrunken, pallid, with an occasional red spot in the cheeks. More or less pain is felt when the bowels are moved or when vomiting is about to take place, which the child manifests by restlessness and a husky whine or cry. Tenderness on pressure usually exists along the track of the colon, and an erythematous rash diffuses from the anus over the buttocks and genitalia, causing so much tenderness that the contact of the irritating discharges excites pain. The mind is, however, rather torpid, the senses not acute, and the attention roused only by strong excitation. The child lies at last in a condition of great exhaustion, indifferent to all surrounding objects, and experiencing the distress which comes from thirst only.

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* "Traité de Climatologie Médicale," vol. iv, p. 317. Paris: Baillière et Fils, 1880.

† "Ptomaines and Leucomaines," by Prof. Victor C. Vaughan.

Rise of temperature takes place with the first disturbance of the intestinal canal, the fever being of the remittent type, with the remission in the morning, usually. In the early morning is the period of greatest depression. With the rise of temperature in the afternoon, the cheeks may be a little flushed, and the countenance, therefore, appear better. The range of temperature taken in the axilla is from 102° to 104° Fahr. in the pronounced cases. The pulse is very rapid and feeble—140 to 160 beats in the minute. The number of discharges may rise to forty or fifty a day, many of them not more than a teaspoonful of fluid. With the progress of the case, there is a rapid decline in weight and strength ; the pulse becomes more quick and feeble ; the respirations grow more and more shallow, and hypostatic congestion and œdema occur ; carbonic-acid poisoning ensues, with a gradually deepening coma, ending in death.

Course, Duration, and Termination.—The ordinary course is prompt in the fatal tendency, or toward cure, the latter being the natural tendency when the child is put under favorable hygienic conditions. The duration of the attack proper is two or three days to one week ; severe cases may terminate in collapse in a day or two. When recovery ensues, the duration of the case is prolonged by the subsequent ileo-colitis. If the prodromic symptoms are included, it may be said that the average cases are from one to two weeks, not including the ileo-colitis or the proctitis, which may prolong the attacks several weeks. The most frequent termination is by exhaustion and death by coma from deficient excretion of carbonic acid and its accumulation in the blood. The cerebral anæmia may be confounded with acute cerebral congestion, and the death attributed, very erroneously, to the latter. Death may happen at the lungs or from failure of the heart.

Diagnosis.—The only disease with which cholera infantum can be confounded is true cholera, but, as the therapeutical indications are the same, it is the less important to be correct.

Prognosis.—A guarded opinion should always be given, as the case may very unexpectedly take an unfavorable turn. The hygienical surroundings influence the prognosis greatly. The number and frequency of the discharges and the readiness with which the symptoms yield to the treatment are important elements in making up a judgment. The constitutional condition, the inherited tendencies, and the aliment available for nutrition, are to be carefully considered. When the child is at the breast, and the supply of milk is abundant and good, the prognosis may be more favorable than if the child has been weaned, and the kind of aliment suitable to the case remains undetermined.

Treatment.—Immediate attention must be given to the aliment. Instead of large draughts of water, the child should suck some pieces of ice. If nursing, the number and duration of applications to the breast must be regulated. The child is excessively thirsty, and is incessant in the demands for nursing. The stomach is quite unable to dispose of it, and it is either soon rejected or passes by the bowels. Once in two, two and a half, or three hours, according to the age, is often enough, and the child should be removed when it has obtained the amount which can be retained by the stomach. If fed by cow's or goat's milk, this should be diluted with lime-water. If they do not agree, owing to an inability to digest the casein, which is the usual difficulty, the best substitute is barley-water, of the density of good milk, to which cream is added in the proportion in which it exists in milk. This combination is a nutritious aliment of the quality of milk, less the casein. Beef-tea is very badly borne in these cases, and the artificial foods prepared for infants are not, in the author's experience, good substitutes for milk. One of the most important remedial agents is the cold bath. The extraordinary temperature range, almost reaching hyperpyrexia, is an important element of danger, causing failure of the heart and paralysis of the brain. The cold bath is the most effective means of combating the fever. The child must be very gently and carefully immersed in water at 95° to 100° Fahr., and the cold water gradually added until the thermometer stands at 85° or 80°, or even 60°, if well borne. The duration of the bath is about ten minutes, and the frequency of their repetition depends on the influence which they have on the temperature. Two or three baths per day are required until the fever permanently declines.

The administration of pure cognac brandy, in a small quantity of very cold water, is an excellent means of checking the vomiting and purging, and of lessening the abnormal heat. From twenty minims to one drachm every two, three, or four hours, according to the age of the subject and the severity of the symptoms, is the proper amount for administration. The opium so much prescribed, and so remarkably beneficial in cholera morbus—a similar state in the adult—is a remedy whose utility is most questionable. In the author's judgment, opium should be given only when the other means used have no effect in restraining the excessive discharges. A most efficient prescription is the combination of bismuth and carbolic acid—ten grains of the former, and one fourth to one half grain of the latter, every two hours. It is best administered with some tincture of cinnamon in an emulsion of gum-arabic. It may be given also with mistura cretæ. Resorcin has been used successfully in the same condition of things requiring carbolic acid. Rhubarb, in doses that are merely astringent, with an aromatic (cinnamon) and an alkali (bicarbonate of potassium), is an efficient remedy, especially in this combination. Infusion of rhubarb, tincture of cinnamon, with some bicarbonate of potassium, makes a disagreeable but extremely serviceable prescription in these cases. Oxide of zinc, oxide of silver, nitrate of silver, are useful in those cases characterized by severe watery purging rather than vomiting. When the vomiting is excessive, and other medicines are rejected, calomel is extremely beneficial, and, indeed, in ordinary cases, it has the first position almost as a sedative to the gastro-intestinal mucous membrane. It must be given in very small doses—one twentieth to one tenth of a grain, every half hour or hour. It may be rubbed up with some sugar of milk and dropped on the tongue. When there is much straining, and especially if there be much mucus, and mucus streaked with blood, passed from the bowels, minute doses of arsenic (from one eighth to one fourth drop of Fowler's solution) with a little opium (one fourth to one half of a drop of the deodorized tincture), every three hours, are very serviceable, and, under the same conditions, very small doses of corrosive sublimate ($\frac{1}{100}$ gr.) may do great good. If the discharges are very profuse, watery, and not restrained by the remedies prescribed by the stomach, enemata of starch and laudanum may be used. Counter-irritation by mustard (the skin very little reddened or irritated), or by means of a spice-bag, or, better, a turpentine-stupe, is beneficial, if not carried too far.

When the algid stage approaches, nitro-glycerin, amyl nitrite, and atropine, may be most effective in reviving the failing circulation.

DUODENITIS—CATARRH OF THE DUODENUM.

Definition.—Catarrh of the mucous membrane of the duodenum may be acute or chronic. As the ductus communis choledochus opens into that part of the canal, the catarrhal process extends up by contiguity of tissue, and hence catarrhal jaundice may coexist with duodenitis.

Etiology.—Climatic changes are very influential in setting up a catarrh of the duodenum. External irritation, if severe and prolonged, will cause hyperæmia and structural changes, just as a severe burn will excite ulceration. Probably the most common cause is indigestible aliment, which passes the stomach unchanged, and the excessive use of starchy, saccharine, and fatty foods, which require for their digestion and absorption the action of the intestinal juices, of the bile, and of the pancreatic fluid.

Pathological Anatomy.—The general description already given applies here. Hyperæmia and œdema occur to a more pronounced extent about the orifice of the common bile-duct, which is so swollen as to encroach materially on the lumen. More or less injection and swelling of the mucous lining of the duct exist to a variable extent.

Symptoms.—The anatomical seat of the inflammation influences, to a great extent, the symptoms. In other cases of intestinal catarrh, diarrhœa is a prominent symptom; in duodenitis, diarrhœa is exceptional, and more or less constipation is the rule. Pain and disorders of digestion are usually present, and jaundice is a prominent symptom.

The pain is felt in the right hypochondriac and umbilical regions, and soreness can be developed by deep pressure over the duodenum. The pain is not usually very acute—the sensation is compounded of pain and soreness, but occasionally severe pain occurs in the hepatic plexus. As in catarrh of the stomach there are occasional attacks of gastralgia, so in catarrh of the duodenum there are occasional attacks of hepatalgia. The paroxysms of severe pain come on gradually, and, after some hours, gradually subside. There is no increased soreness during the existence of the pain or subsequently.

There may or may not be present gastric catarrh, as well as duodenitis. The distress caused by the presence of food is felt about three hours after it has been taken, and is usually referred by the patient to the seat of the disease. The starchy and saccharine elements of the food undergo fermentation, and hence, in about three hours after they have been swallowed, the formation of flatus begins, the small intestines become distended with gas, and some pain, due to the stretching of the bowel, is felt about the umbilicus. From the third to the seventh day jaundice appears. It is usually announced by a coated tongue, fetid breath, and yellowness of the conjunctiva, headache, stupor, and hebetude of mind (cholæmia), with depression of spirits. The yellowness extends, and in a short time the jaundice is universal. The absorption of bile is coincident with swelling of the common duct, and entire absence of bile in the intestinal canal. The stools now have a pasty consistence, a slate-color, and fetid odor. Gas, discharged previously, had but little odor; after the jaundice, it has the same fetid character as the stools. The urine is thick from excess of urates, and of a deep-brownish color from presence of bile-pigment. When the jaundice has attained the maximum, there are complete anorexia, nausea, sometimes vomiting of food, mucus, sero-mucus, and constipation, although diarrhœa may occur; but instead of jaundice there may be merely the condition of biliousness. The temperature is slightly elevated—99·5° Fahr. in the morning and 100° to 101° Fahr. in the evening. The pulse corresponds.

Course, Duration, and Termination.—The disease is self-limited, and, if permitted to pursue its course uninterrupted, will last two or three weeks, leaving the patient much debilitated. In malarious districts this malady is exceedingly common, and may be intimately associated with malarial infection. The chronic form of duodenitis is essentially the same in respect to clinical history and characters, except as to duration and violence of the symptoms, as the acute form. The duration of the chronic form may be several months. The late researches of Charcot and Legg have demonstrated that long-continued obstacle to the outflow of bile leads to structural changes in the liver. The termination of uncomplicated duodenitis is in health. The acute is apt to pass into the chronic form, and the latter to affect the hepatic parenchyma in the manner to be hereafter described. Hepatic colic is also one of the results of this disease.

Diagnosis.—Duodenal catarrh may be confounded with gastric catarrh, with hepatic colic, and with diseases of the liver proper, accompanied by jaundice. As respects gastric catarrh, the differentiation is to be made by reference to the seat of pain and soreness, the time when the distress from the presence of food comes on, the occurrence of flatulence with bowel-pain, and especially the appearance of jaundice at a certain time after the beginning of the symptoms. Duodenal catarrh is separated from hepatic colic by the following signs: In the latter, the pain comes on suddenly after some pain and soreness in the region of the gall-bladder, and radiates from this point over the abdomen; the pain is so intense as to produce a cold surface, a weak pulse, great depression, and incessant vomiting; the pain suddenly ceases, and there is complete relief, except some local tenderness; jaundice follows these symptoms, but disappears in a few days, leaving the patient well; and a gall-stone may be found in an evacuation a few days after the attack. Hepatalgia is a neuralgic attack, occurring suddenly, and limited to the hepatic plexus. It ceases suddenly, leaving the patient well, and the only interference with function is during the existence of pain. Its duration is but a few hours.

Treatment.—The first point is regulation of the diet. The diet should be restricted to those substances convertible into peptones in the stomach, as milk, whey, buttermilk, eggs, animal broths, and all saccharine, starchy, and fatty constituents should be avoided. Fresh meats, game, poultry, and fish, without butter or fat, are admissible if the stomach is equal to their digestion. The most rapid progress can be made by adhering to an exclusive diet of milk, and, as there is complete anorexia, this is usually not difficult. The hyperæmia of the duodenal mucous membrane is relieved by saline laxatives, by the Saratoga, Carlsbad, or Vichy waters, by Rochelle salts, but especially by phosphate of soda, which should be given in drachm-doses about four times a day. Other remedies, acting similarly, are sulphate of magnesia and bitartrate of potassa. The general principle is to use remedies which will promote an outward diffusion, and thus relieve the congestion and œdema of the mucous membrane. Small doses of calomel ($\frac{1}{6}$ grain to one grain) may be highly useful as a sedative and antiferment, and acting in the same way are bismuth, oxides of silver and zinc, but especially the purified black oxide of manganese. Active cholagogues, as the resin of podophyllin, rhubarb, aloes, etc., are to be avoided on account of the irritation which they induce. Excellent results are had from the benzoates—the benzoates of sodium and ammonium, especially the latter. Benzoate checks fermentation in the duodenum, lessens stasis in the portal system, and promotes elimination by the kidneys. To rouse the liver—a favorite phrase—is out of place here, since the obstacles to the outflow of bile are merely mechanical. When malarial infection coexists, quinine is indispensable to restore health. Without any complication of malaria, quinine has a good effect, and hastens the disappearance of the jaundice. When the bile enters the intestine and the intestinal digestion is restored, the jaundice may still linger. Diuretics and purgatives may then be employed to remove the last traces of bile-pigment.

ILEITIS—ILEO-COLITIS—CATARRH OF THE ILEUM AND OF THE ILEUM AND COLON. ACUTE DIARRHŒA; CHRONIC DIARRHŒA.

Definition.—Ileitis is a catarrh of the ileum, either acute or chronic; ileo-colitis is a catarrh involving both parts—the whole extent of the ileum and the cæcum and ascending colon. This may also be either acute or chronic. The disease is frequently denominated diarrhœa, from a single symptom.

Causes.—The causes already given for other forms of intestinal catarrh are equally true of this form. The two great factors are improper and indigestible food and the summer temperature. An attack may be brought on by exposure to cold and damp air when in a perspiring state. The sudden arrest of cutaneous transpiration precipitates a vicarious duty on the mucous membrane, with the effect to induce a general hyperæmia of the ileum and colon. As respects children, the causes in operation to produce ileo-colitis are the same as those which bring on cholera infantum.

Pathological Anatomy.—In this variety of intestinal catarrh, the morbid anatomy has the special feature of enlargement of the agminated patches, which are most abundant and most highly developed in the lower ileum. The condition of the epithelium, of the villi, and of the glands, has been described. Sufficient emphasis has, probably, not been put on the tendency of the swollen glands to ulcerate. In the acute cases the orifices of the solitary glands are here and there eroded; but in the chronic cases considerable ulcers form. These changes are different in character and very different in extent from those which take place in typhoid.

Symptoms.—The acute form of ileitis or ileo-colitis sets in with some chilliness and general *malaise*, followed by feverishness. Pain in the abdomen, usually about the umbilicus, is felt, and then loose evacuations begin. The number of stools each day varies with the character of food and the extent of the disease, especially in the colon. It would be a mistake to suppose that the diarrhœa is due solely to an irritation of the affected portions of the mucous membrane by the particles of aliment reaching them. Considerable transudation occurs as one result of the hyperæmia: cast-off epithelium, young cells, and minute sloughs mix with the serum, and constitute no small part of the stools discharged. Besides, the chyle imperfectly prepared for absorption, and hurried down the canal by the increased peristalsis, and the fatty, starchy, and saccharine constituents of the food, fermenting instead of digesting, unite to form the liquid discharges characteristic of ileo-colitis. As might be expected, there is little fecal matter proper, and the stools have a yellow or greenish-yellow color, and, if the evacuations have been very copious, they may be whitish, like the "rice-water" discharges. In children the stools have a somewhat different character, owing to the presence of casein, which presents an appearance of putty, or the casein occurs in small, irregular masses. Very often the stools have a bright-green color, or become green on exposure. Just before the evacuation considerable pain is experienced, and, in children, nausea and vomiting also. The pain is usually increased by pressure, and soreness is developed at any time by deep pressure. As gases are freely generated in food decompositions, the intestines are often suddenly distended, giving rise to pains as of flatulent colic. Borborygmi are more or less present. It is a curious fact that mental depression is a very constant condition in cases of ileo-colitis when there is abundant production of gas. The digestion and assimilation of food being almost arrested, and great waste taking place by the intestinal mucous membrane, it is obvious that the organism must lose ground rapidly. The subcutaneous fat disappears; the muscles shrink and lose their contractile energy; the skin becomes dry, sallow, and wrinkled; the action of the heart is weak, the pulse small and feeble; the urine is acid, high-colored, and burning. Children affected with summer diarrhœa, and having from three to six evacuations a day and vomiting occasionally, rapidly emaciate, are reduced to a skeleton in fact. In the adult the chronic form is known as "chronic diarrhœa," in which, as is well known, the wasting of the tissues of the body proceeds to the lowest point.

Course, Duration, and Termination.—In the simplest cases of catarrh of the intestine, due merely to an unusual accumulation of fæces—crapulous diarrhœa—the looseness of the bowels is conservative, an effort of nature to be encouraged rather than restrained. In mild, uncomplicated cases the tendency is to recovery in a few days, but in the severe cases the duration may be several weeks. In the chronic form the duration is indefinite. The acute runs insensibly into the chronic form, and there is no well-marked distinction, except the element of time.

Diagnosis.—The distinctions to be made are between duodenal catarrh and catarrh of the rectum (proctitis). In children, ileo-colitis is to be distinguished from cholera infantum. In duodenal catarrh there is little or no diarrhœa, and jaundice appears in a few days, symptoms entirely different from ileo-colitis. In proctitis the stools may be normal, or occur as scybala. There are straining, heat, and irritation about the rectum, and the discharge of mucus, and mucus and blood. In children, ileo-colitis is frequently mistaken for and called cholera infantum. The latter is a disease of sudden onset, characterized by choleriform symptoms and a duration of a few days or few hours only. The character of the discharges is essentially different; in ileo-colitis they contain casein, yellowish or greenish liquid matter, spinach-colored masses; whereas, in cholera infantum, they are serous in character, colorless, like the so-called rice-water evacuations, and do not leave anything but a stain on the napkin.

Prognosis.—In acute diarrhœa, under good hygienic conditions, the prognosis is favorable. In children, summer diarrhœa is amenable to treatment or not, according to the condition in life, and the ability of parents to provide the necessary means. When ileo-colitis has become chronic, and is not readily amenable to the treatment, the prognosis is grave. In adults, for chronic diarrhœa, which has long existed, the prognosis must be guarded.

Treatment.—In simple acute catarrh relief is afforded by a pill of opium and camphor. When the evacuations are numerous and profuse—summer diarrhœa, for example—the most efficient treatment is the combination of a mineral acid (muriatic or sulphuric) with tincture of opium. Carefully managed, the same remedies may be administered to infants. Sometimes alkalies agree better. Sodium bicarbonate can be given with or without bismuth in chalk-mixture. Alkalies, however, merely neutralize acids, but the mineral acids check the fermentation on which the production of acid depends. When the discharges are greenish ("chopped spinach"), the combination of arsenic and opium is highly efficient—for example, one drop of Fowler's solution, and one drop or less of the deodorized tincture of opium. When there are retained matters, the presence of which excites irritation, an emulsion of castor-oil, with two or three drops of turpentine and some tincture of opium, is very advantageous. In the more chronic cases, or after the acute symptoms have subsided, sulphate of copper with a little opium is an admirable remedy—from one thirtieth to one twelfth of a grain of copper sulphate, and one fortieth to one sixth of a grain of morphine, according to the age of the subject. Other astringents, metallic and vegetable, may be employed under the same circumstances. For children, bismuth is probably the best astringent. Resorcin has been found to be an effective remedy. Ergot has now and then proved a valuable expedient, and in some cases, especially of the more chronic kind, cubeb, eucalyptus, and others of this group, have rendered important aid. Regulation of the diet is even more important than the use of medicines. The starchy, fatty, and saccharine articles of food are highly objectionable, and should be omitted entirely, as already advised. The same plan of diet suggested in previous articles is applicable here, and need not, therefore, be repeated.

TYPHLITIS.—INFLAMMATION OF THE CÆCUM.—CATARRH OF THE CÆCUM.

Definition.—The term typhlitis is restricted to an inflammation of the cæcum and its appendix. Perityphlitis is an inflammation taking place in the loose connective tissue on which the cæcum rests. Although the seat of the lesion and its nature are very different, it is necessary, because of their intimate relations, to consider them together.

Causes.—Besides the causes of catarrh of the intestines already sufficiently set forth, there are special conditions affecting the cæcum. The anatomical position of this organ as a receptacle for the small intestine, the arrangement of its muscular elements, the abundant folds of mucous membrane when empty, and its immense capacity when filled, are properties necessary to its function, but at the same time causes of disease.

Pathological Anatomy.—Catarrh of the cæcum may exist as a mere catarrhal affection of the mucous membrane, with the changes in the epithelium, in the solitary glands, and in the vessels already described; or as a localized inflammation, usually from the presence of a foreign body, terminating in ulceration; or as an inflammation of the cæcum in general, with a more intense action about the ileo-cæcal valve, and implication with thickening of the submucous connective tissue, causing stenosis. The second or ulcerative form of catarrh of the cæcum will be described hereafter under ulcers of the intestinal canal. The last-named variety remains for consideration. The ileo-cæcal valve being more exposed to injury than any other part of the cæcum, owing to its position and office, is more liable to be invaded by disease. When a catarrh of the cæcum exists, especially the chronic form, the hyperæmia and swelling are more decided in the neighborhood of the orifice. An extension of the inflammation to the submucous layer occasionally takes place, the connective tissue undergoes hyperplasia, a permanent increase of thickness results, and stenosis is an ultimate effect of the changes. It is only in the chronic form that such thickening and stenosis can occur.

Symptoms.—There are two forms of catarrh of the cæcum—the acute and chronic. Of the acute variety there are various grades in the severity of the cases, but two are sufficiently defined to require attention—the mild and the severe. In the mild cases, uneasiness, followed by pain and soreness, is felt in the right iliac region, extending up along the course of the ascending colon. On palpation, tenderness is found to exist in this region, and laterally just above the crest of the ilium. The more decided the pressure, the more distinct the pain. Early, and before the inflammation has extended beyond the mucous layer of the cæcum, the decubitus and the sitting posture are characteristic—the body is turned toward the right side, and is flexed somewhat to relax the muscles on the right lateral plane. Additional soreness is experienced when the body is held erect, or straightened out in bed. With the first symptoms there may be some accumulation of fæces, and the cæcum and ascending colon may be distinctly bulging and prominent, so that they may be recognized on inspection; but in the mild cases there is no impaction, properly speaking, but on careful palpation the outline of the bowel can be made out, feeling rather soft and dough-like. The bowels are usually constipated, for catarrh of the cæcum seems to affect the muscularis, impairing its contractile energy, or there may be an appearance of relaxation by reason of an accumulation in the sacculated periphery of the bowel—leaving a central cavity along which the liquid contents of the small intestines may pass. The author has seen several examples of this, and so important is the recognition of the condition that he now desires to emphasize the fact. During the development of these local symptoms, the system partakes in the disturbance. The attack sets in with general *malaise*, some feverishness, a coated tongue, loss of appetite, nausea, and not unfrequently vomiting. In the severe cases the symptoms are increased in severity in all directions. The local pain, tenderness, and swelling are greater, there are impaction of fæces and no movement. There are decided fever, considerable restlessness, nausea, and vomiting. The vomited matters consist at first of the contents of the stomach, then of the duodenum with much bilious matter, and ultimately, if the impaction persist, of matter that has somewhat the odor of fæces. With the development of the case there occurs great depression of the powers of life, the face becomes pinched and anxious, the skin covered by a clammy sweat, the pulse small and rapid, the action of the heart weak. Peritonitis is finally developed by contiguity of tissue, or by the bowel giving way at some point, weakened by ulceration. The subsequent history is then the history of peritonitis.

In the chronic cases, which may succeed to the mild acute, or, as is much more common, develop slowly by the operation of the ordinary causes of intestinal catarrh, the symptoms are those of intestinal indigestion. There is uneasiness in the region of the ileo-cæcal valve, flatus is felt passing the orifice, and the patient is often conscious of the difference in density, whether gas, liquid, or solid, of the materials passing the orifice. The bowels are confined and rather difficult to move. When the actions are free, semi-solid, and unirritating, the patient has a keen sense of relief. Rarely, on careful palpation, induration, not hard like that of scirrhus, but doughy, can be made out. A comparatively empty state of the large intestine and distention of the small intestines can usually be ascertained; in that event the lateral portions of the abdomen are rather flat, and the central part around the umbilicus is prominent.

Course, Duration, and Termination.—The mild form of acute catarrh of the cæcum, if properly managed, is readily cured in a week or two. The severe form may terminate by acute peritonitis within a week, or be relieved, and all pain and tenderness subside, within two or three weeks. Very frequently entire recovery does not ensue, but the case passes into chronic catarrh, the duration of which is very indefinite; or, associated with perityphlitis, a chronic abscess may continue in a torpid state for some time, or a fistulous communication be established with the exterior. Abscess of the liver is a frequent result of catarrhal inflammation and ulceration of the cæcum.

Diagnosis.—It is often extremely difficult to distinguish typhlitis from perityphlitis or from occlusion of the bowel by other kinds of obstruction. The points of difference between typhlitis and perityphlitis can be better understood after the study of the latter, and are therefore reserved. Typhlitis in the mild form is distinguished from other affections of the bowel by the local pain and soreness, by the fullness without impaction; in the severe form, the symptoms of obstruction are the same as in other kinds of occlusion, but the local pain and the distinct enlargement of the bowel indicate the existence of an inflammation and fecal obstruction of the cæcum. In these affections, the decubitus of the patient is an important aid to diagnosis. Chronic catarrh of the cæcum is recognized by the locality of the distress. As cancer of the cæcum behaves in the same way in the early stage of its formation, there may be no means of differentiating ; but, in the progress of the case, the growth of a nodulated tumor, the progressive increase in the pain and obstruction, and the development of a cachexia, are sufficient to indicate the nature of the affection.

Prognosis.—In the simple form the prognosis is favorable ; in the severe form it is grave, although recovery will ensue in a large proportion of the cases if the management is judicious. In the chronic form, when the connective tissue has become thickened, the prognosis as to cure is unfavorable.

Treatment.—In the treatment of acute typhlitis all active purgatives must be avoided. If there is but little feverishness, and the local tenderness is slight, saline laxatives may be administered from the beginning, in small doses at short intervals, to induce liquefaction of the contents of the bowel. The hyperæmia is lessened by the same means. When free discharges are obtained in this way, the canal should be kept quiet with opium for a few days. The most efficient and, at the same time, safe laxative is sulphate of magnesia. It is a curious fact that this salt will be retained when other salines are rejected by vomiting. Rochelle salts may be used as a substitute when Epsom salts is not suitable. Different management is required in cases of typhlitis with impaction and arrest of the intestinal movement. If there be fever and much tenderness, no attempt should be made to relieve the bowels by purgatives of any kind. It is in this condition of affairs that opium in some form, especially in the form of the hypodermatic injection of morphine, is so serviceable. The patient should be kept thoroughly under the influence of the narcotic. It is better to combine atropine with the morphine, for greater security and increased therapeutical power. No absolute rule for quantity can be laid down, but the decided effects of morphine, as shown in the state of the pupil, the pulse, the respirations, and the somnolence, should be steadily maintained. The fullest curative power of morphine is obtained from a quantity strictly within the limits of safety, and hence no risk need be had to obtain the best results. As a guide to the administration, it may be stated that one fourth of a grain of morphine and $\frac{1}{120}$ grain of atropine is enough for the first dose in an adult, and subsequently one eighth of a grain of morphine and $\frac{1}{240}$ grain of atropine may be given every four, six, or eight hours, according to the effect. If there be any reason, moral or physical, which prevents the hypodermatic administration, the next best mode is the rectal injection of the tincture of opium. As respects the quantity, the rule above given is proper; it is the degree and constancy of the effect which determine the amount. If the rectal injection is objected to, or the organ is intolerant, opium must be administered by the stomach. The best preparation is the deodorized tincture, and, to secure uniformity in action, the preparation made after an essay of the opium is altogether preferable. This corresponds in strength to laudanum: sixty drops may be the first dose, and twenty drops every two, three, or four hours succeeding, the quantity to be determined by the effects, as already insisted upon. The administration of the opium is to be continued until the bowels are moved spontaneously, or until the inflammatory action—the fever and local tenderness—subsides. The effects may be maintained for several days, for a week or more. As soon as the tenderness subsides, the saline laxative may be then given, in the cautious way already advised —a teaspoonful of Epsom salts in two ounces of water every three hours. With the subsidence of the local tenderness and heat, the quantity of opium can be slowly reduced and the interval between the doses lengthened. If the vomiting be persistent, it may be relieved by milk and lime-water (three parts to one), carbolic acid (gr. ss. in cherry-laurel water), hydrocyanic acid (♏iij), iced champagne, pellets of ice, etc., but when the hypodermatic injection is practiced vomiting is a much less pronounced symptom. In robust subjects, and in all cases not characterized by great debility, leeches should be applied at the seat of tenderness, and in numbers according to the state of the patient —from two to ten to be allowed to fill and drop off, and the bleeding be then arrested. Good effects are obtained from counter-irritation by mustard, followed by fomentations of turpentine, or turpentine stupes, and hot poultices, when heat applications are useful. According to the author's observation in these cases, the external application of ice —in the form of an ice-bag—is more efficient than warm applications. In the severe cases of typhlitis, when the time has arrived for attempts to remove the impaction, the action of the saline laxative may be aided by irrigation of the bowel. It is now known that by this method the bowel may be filled with fluid up to the ileo-cæcal valve. Accordingly, repeated efforts by enemata of warm soapsuds should be made to soften the masses of hardened fæces which so effectually block the canal. The use of a long rectal tube to convey the fluid beyond the sigmoid flexure facilitates the operation materially. If impaction has existed for several days, care must be used in distending the bowel, for it may yield to the pressure, softened it may be by an inflammatory process involving all the layers. Lately it has been found that irrigation of the stomach, as practised in stomachal diseases, is an effective means of relief in cases of impaction of the intestine.

INFLAMMATION OF THE APPENDIX VERMIFORMIS.—The usual cause of inflammation of the appendix is the lodgment of an intestinal concretion, grape-seed, or other foreign body.[*] Cases of inflammation, apparently catarrhal, do, however, rarely occur, and very serious symptoms quickly arise by extension of the disease to the peritoneal layer. The symptoms are the same as those of the severe form of typhlitis, with some important exceptions to be presently detailed. The appendix differs from the cæcum in that it has an entire peritoneal investment, and is freely movable except at its point of attachment to the cæcum. In some subjects the appendix is two inches in length, and hence dips down into the iliac region to the pelvis, and reaches almost or quite to the bladder. When, therefore, an inflammatory process occurs in it, the tenderness and pain are felt in the iliac region as low down as Poupart's ligament, and not in the cæcum. When typhlitis exists, the appendix becomes involved, but death may and does frequently follow from disease of the appendix, without the cæcum being implicated. When, therefore, this form of typhlitis occurs, besides the symptoms already set forth, there is pain in the groin, extending down the course of the anterior crural, and through the hip. The tenderness is usually exquisite, and the slightest attempt at palpation gives the patient great dread. The thigh is flexed on the pelvis, and all attempts to extend it cause great suffering. There is no fecal tumor such as is found in typhlitis with impaction, and the bowels are not affected, but all intestinal movements, as the passing of gas through the ilio-cæcal valve, cause pain. Peritonitis, much more readily than in affections of the cæcum, occurs in inflammation of the appendix. It is often entirely local, and, adhesions forming, the morbid action is cut off from the general cavity of the abdomen. This is one of the modes by which fecal abscesses are formed. This subject and peritonitis are properly topics for future consideration.

PERITYPHLITIS.—As the term indicates, this is an inflammation of the tissue about the cæcum—really, of the connective tissue in which the cæcum is in part imbedded. This may arise spontaneously—an inflammation of the connective tissue—by the ordinary causes of such inflammation, especially trauma. It may be caused by the extension of inflammation from the cæcum, by perforation of the cæcum. Its special tendency is to suppuration. When well developed there is a hard, brawny swelling felt above the crest of the ilium, extending back into the lumbar region. There is not usually acute pain, but a feeling of weight, soreness, with paroxysms of subacute pain, extending into the hip, thigh, and abdomen. There is no necessary interference with the bowel, unless typhlitis and perityphlitis coexist. The development of the swelling is comparatively slow, but it attains considerable dimensions. Suppuration is preceded by an increase of the local distress ; when it has actually taken place, the tension and throbbing diminish for a time, to increase again as the pus nears the surface. The formation of matter is attended by the usual constitutional symptoms.

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[*] See cases reported by the author in his paper on typhlitis, in the "American Journal of Medical Sciences," October, 1866, p. 351.

The treatment of perityphlitis is the same as that of typhlitis, except as regards the special attention given to the bowels, and entirely the same if the two maladies coexist. When pus forms in perityphlitis, and when a sero-purulent collection is formed by a limiting inflammation, in inflammation or perforation of the appendix, there arises the surgical question of an operation for the evacuation of the matter. By the use of the aspirator, the question of suppuration may be early determined. It is no doubt sound practice to pursue the method of Buck, and procure the evacuation of pus by a sufficient opening for free drainage.*

CATARRH OF THE RECTUM.—PROCTITIS AND PERIPROCTITIS.

Definition.—Catarrh of the rectum is known as proctitis. In the mild form it is the simplest kind of dysentery. In the severe form, as in the cæcum, there may be impaction of the colon at and above the sigmoid flexure. The two forms correspond to the same conditions in the cæcum. The analogy becomes the more complete by reason of periproctitis—an inflammation of the connective tissue about the rectum.

Causes.—Proctitis arises chiefly from constipation. Prolonged retention of hardened fæces sets up an irritation for their expulsion. It is also caused by cold and dampness combined, especially sitting on the ground while in a perspiring state. Distention of the hæmorrhoidal vessels, by obstructive disease of the liver, as in cirrhosis, is an occasional cause, but the disease then is quite masked by the more important results of the cirrhosis. The habitual use of stimulating enemata and of aloetic purgatives is a fruitful source of proctitis.

Pathological Anatomy.—The alterations of structure are the same as those already described.

Symptoms.—There are an acute and chronic form, the symptoms of which differ in degree merely. The acute variety exists in two forms, the mild and severe. In the mild form of proctitis, the patient experiences a sense of uneasiness in the rectum—a burning, with desire to go to stool. There is much straining, and only mucus passes. The sphincter ani is in a constant state of spasm. Immediately after the passage of some mucus, there is felt considerable burning pain, and a sensation as if something remained, so that the patient returns again and again to the close-stool, and as before passes only some mucus or mucus mixed with blood. This condition is called *tenesmus.* The pain radiates from the rectum to the hips and back, and a feeling of depression and anxiety, and often of nausea, accompanies it. The colon is distended above the sigmoid flexure, but only some hard, roundish masses of fæces, known as scybala, descend occasionally. In the severe form all of these symptoms are intensified, the pain is very acute, intensely burning, and widely diffused. The straining is violent, and prolapse of the mucous membrane takes place, the sphincter ani closes over it spasmodically and the protruding portion becomes excessively painful, purplish, and bleeding. The mucus discharged is mixed with blood, and sometimes considerable hæmorrhage occurs in consequence of the yielding of a vessel. The colon above is impacted with hardened fæces, and its outlines can be distinctly traced by palpation. In the severe form of proctitis there is usually some constitutional disturbance—some feverishness, headache, and general muscular soreness. The neighboring organs sympathize with the rectum. In the female, the menstrual flow may occur, and, in both male and female, strangury comes on, and with the straining at stool there is simultaneous straining at the passage of urine. The long-continued distention of the colon induces an irritation of the mucous membrane; a catarrhal process is set up for the expulsion of the accumulated fæces, but the muscular layer, over-distended, becomes paretic and is incapable of any energetic action; the inflammation extends and ultimately the peritoneum becomes involved. The progress of these structural changes is manifested objectively by an increasing tenderness along the track of the descending colon, and finally by an extension of the inflammation to the adjacent connective tissue, the formation of a tumor, terminating in an abscess. In the cavity of the pelvis a similar process may take place, the inflammation of the mucous membrane extending by contiguity to the layers of the bowel successively, and at length involving the neighboring connective tissue. The chronic form of proctitis presents nearly the same features. There are usually accumulations of scybala in the sacculated periphery of the colon, but the bowels may be confined or relaxed. The relaxed stools contain a good deal of mucus, and are highly offensive by reason of the decompositions which have ensued in the descent along the colon, and the scybala are coated with mucus. Instead of ordinary mucus, the matter now discharged contains purulent elements—muco-pus—and ultimately becomes entirely purulent in the rectum. Ulcerations ensue, sloughs separate, and hence the stools contain the *débris*. The nerves become somewhat accustomed to the irritation of their terminal filaments in the mucous membrane, and therefore the reflex incitement to tenesmus is much less. There are, therefore, less straining, less acute pain, but the stools are more unhealthy.

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* "New York Medical Journal," vol. ii, p. 38. Numerous cases have been reported of some foreign body discharged by a fecal abscess. Hence, the need of a free opening.

Course, Duration, and Termination.—The mild form of catarrh of the rectum has a natural tendency to cure in from four to eight days. The bowels act freely, the colon is emptied, and the tenesmus ceases. In the more severe cases, although a spontaneous cure may result, yet there is great danger of peritonitis, or periproctitis and abscess. When the latter forms, it tends to discharge alongside the rectum, resulting in fistula usually, or into the vagina or neighboring organs, forming various kinds of fistulæ. The duration of the severe form is determined largely by the character of the treatment. The chronic form is obstinate, and pursues a uniform course leading to extensive ulceration, sometimes perforation and peritonitis, or cicatrization and permanent encroachment on the lumen of the bowel. Thrombosis of the inferior hæmorrhoidal veins, with subsequent formation of hepatic abscess by deposit of emboli, is a not uncommon result. These changes are all promoted by the fermentations occurring in the rectum, the products of which are highly irritating and offensive.

Diagnosis.—The symptoms of acute proctitis are so distinctive that the diagnosis is made by them. In women, irritation of the rectum and tenesmus are produced by retroversion, especially of the gravid uterus. A vaginal exploration may be necessary to determine the position of the womb : if the symptoms persist after the malposition is rectified, then it may be justly assumed that disease exists in the rectum. In women, the eversion of the rectum through the sphincter ani is so readily performed that the nature of the case may be determined by ocular inspection. Exploration of the rectum may be necessary to differentiate between ulcer of the rectum and chronic proctitis. Many of the symptoms may be due to hæmorrhoids ; an examination should be instituted whenever doubt exists. The author has known of two instances in which fecal accumulation and catarrh of the rectum were mistaken for scirrhus, and an unfavorable prognosis given.

Prognosis.—A favorable termination may be predicted in every case of acute proctitis, unless implication of the peritoneum, perforation, or periproctitis has occurred. When peritonitis has arisen, the prognosis is extremely unfavorable if it is general, especially if from perforation, but is less gloomy when limited by adhesions. In the suppuration which then ensues, the resources of the organism are severely tried ; in suppuration from periproctitis low down, although the strength may be much reduced, a fatal result is very rare ; but in these cases the local condition may be a mere expression of a dyscrasia, as tuberculosis, and they are to be estimated accordingly. In chronic proctitis the gravity of the case is increased by accidental and consequential complications. The existence of cirrhosis is unfavorable, as it keeps up a constant over-fullness of the inferior hæmorrhoidal veins. Obstructive cardiac and pulmonary diseases act in the same way, though not so directly. The more changed the mucous membrane is in structure, the more extensive and deep the ulcerations, and the greater the hypertrophy of the muscular layer, the more serious the case. A very important complication is thrombosis of a hæmorrhoidal vein, with detached emboli, and subsequent multiple abscess of the liver. When this condition of things exists, the gravity of the case is vastly increased.

Treatment.—Unless impaction is complete, and the peritoneal layer of the bowel implicated, the first duty to be done is to empty the colon of its retained fæces. It is a most serious mistake in treating acute catarrh of the rectum (dysentery), and one frequently made, to employ astringents and anodynes with a view to quiet the straining at stool. When the bowels are freely evacuated, little remains to be done in the ordinary cases. As already indicated, under similar conditions, there is no laxative so safe and efficient as Epsom salts. It should be given in solution with dilute sulphuric acid—ʒ ij of sulphate of magnesia and ♏ xx of dilute sulphuric acid in two ounces of water every two hours until the bowel is emptied. The straining at stool and the pain may be then promptly arrested by the hypodermatic injection of morphia, or by enemata of tincture of opium in starch-mixture, or by opium in some form by the stomach. In the severe cases, the action of Epsom salts may be aided by irrigation of the bowel. A considerable quantity of warm water should be slowly injected, and retained as long as possible to soften the hardened fæces, and successive injections should be practiced at short intervals. These lavements are useful in allaying the excessive irritability of the mucous membrane. Other salines may be used, but none are so effective as the Epsom for this particular purpose. Enemata of emollients may be used instead of hot water—for example, infusion of flaxseed, of elm, of camomile, etc.—but they are really less efficient, because they are less solvent of the fæces. Various purgatives, notably castor-oil, have been used to dislodge the impacted fæces, but they do not establish an outward diffusion to diminish congestion of the mucous membrane, which is the important action of the salines. In the severe form of proctitis, in robust subjects, and even in the weakly, leeches should be carefully applied around the margin of the anus. If there be much tenderness, an ice-bag should be applied over the descending colon, or warm fomentations, as already advised, for corresponding states. In chronic catarrh of the rectum, the diseased membrane can be reached directly, and the treatment should, therefore, be largely topical. Solutions of tannin (℈ j—℥ iv), of fluid extracts of hydrastis and rhatany, and of other vegetable astringents, are effective local applications if there are no solutions of continuity, but, if ulcerations exist, the most efficient topical application is nitrate-of-silver solution—four grains to a scruple, to an ounce of water. This should be injected through a tube carried up to the sigmoid flexure. Next to silver nitrate is the sulphate of copper, but this must be used very cautiously. It is important in these cases to maintain a soluble state of the bowels. When constipation occurs, the congestion of the mucous membrane is increased, and *vice versa*. Hardened fæces irritate in passing the inflamed membrane. As fermentation, producing most unhealthy products, takes place in the rectum, morning and evening enemata of hot water should be regularly used. They give great comfort, and contribute materially to the cure. The wasting caused by chronic catarrh of the rectum demands the use of the most nutritious food. Cod-liver oil is highly serviceable as food and medicine. If the digestion is feeble, it should be aided by the mineral acids and pepsin, and by nux vomica. Although medicines by the stomach occupy an inferior position in the treatment of this malady, excellent results are obtained from the use of minute doses of corrosive sublimate (one fortieth grain *ter in die*), of arsenic (two drops of Fowler's solution *ter in die*), or of sulphate of copper (one sixteenth grain *ter in die*) ; and when there is much mucus produced, cubeb, eucalyptus, and hydrastis act favorably.

CROUPOUS OR MEMBRANOUS ENTERITIS.

Definition.—By this term is meant an inflammation, subacute or chronic, occurring periodically, and characterized by the formation and discharge of membranous shreds or casts.

Causes.—This is a disease of adult life chiefly ; it is rare in childhood, and does not appear after forty-five. The female sex is more liable than the male ; and nervous, hysterical, and hypochondriacal subjects are more subject to it than are other types. A peculiar state of the nervous system seems necessary to its production. Membranous enteritis occurs by extension of the diphtheritic process downward, and false membrane also forms in infective dysentery, but the disease under consideration is a distinct affection. It has been attributed to the ordinary causes of catarrh of the intestines—especially to irritants, as drastic purgatives, coarse food, etc.—but such agencies can act only as exciting causes.

Pathological Anatomy.—Besides the exudation of diphtheria and of infective dysentery, deposits of a white or grayish-white color, flaky or membranous, and firmly adherent, have been found on the mucous membrane of the ilium and colon. Occurring first in isolated patches, the membrane extends laterally along the mucous folds in the small intestine, and in the colon upon the ileo-cæcal valve and the folds of the sigmoid flexure (Leube). In other cases (Sir James Simpson) papular and white vesicular eruptions have been found, but no flaky membrane or casts adherent to the mucous membrane.

The membrane as passed has been carefully examined microscopically and chemically by Da Costa,* whose memoir on this disease is by far the most important contribution which has been made to our knowledge of the subject. The shreds, casts, or membranous masses, consist of "a transparent, amorphous, basement substance, here and there indistinctly fibrillated, and having imbedded in it granules, free nuclei, and small, shriveled, irregular, and rather granular cells." Chemically, this material has the same reactions as mucus (Da Costa) —a fact which might *a priori* be expected, since this false membrane is nothing more than solidified mucus, the granules, free nuclei, and granular cells found in it being remains of mucus-cells which escaped entire destruction in the process of solidification. The mucous membrane of the rectum, in a case examined by Da Costa, was intensely injected.

Symptoms.—The attacks are announced by a feeling of soreness and distention of the abdomen, and constipation. There is no fever, the hands and feet are cold and moist, and the general condition that of depression, in which the mind participates. Before, indeed, any local manifestations of disease, there are apt to be attacks of hysteria or hypochondriasis, and the subjects of this disease are nervous, excitable, neuralgic. The pains have the colicky character, are felt around the umbilicus chiefly, and are exceedingly severe and depressing. They continue for a half hour, for an hour or two, and even longer, and, after a variable interval of some hours' duration, occur again. Thus, during the twenty-four hours, there may be six or more paroxysms. The distress does not cease with the subsidence of the acute pain : a feeling of rawness and soreness remains, and the abdomen is so sensitive to pressure that peritonitis may be suspected. Very considerable tenesmus exists, and more or less mucus, with or without blood, is passed, as in acute catarrh of the rectum. There may be several loose evacuations a day, or the bowels may be confined. After several days of suffering, there will be discharged, with great pain and tenesmus, shreds of membrane or cylindrical casts of the bowel. Great relief is experienced. The soreness subsides, the distention lessens at once, and the tenderness diminishes. The patient is left in a condition of great debility and much emaciated, for during the paroxysm there is complete anorexia, and sometimes vomiting, so that but little food is taken. The paroxysms are rarely single ; in a week or two, or after several months, there is a renewal of the same experiences. In one of the author's cases there were paroxysms several times a week for three weeks, the patient passing an almost incredible quantity of false membrane. The same woman, in an attack three years before, had a succession of paroxysms for six weeks, and was so reduced that her life was despaired of. During the interval of three years there were no paroxysms, but she suffered from constant troubles of digestion. In the cases related by Da Costa, disorders of digestion continued and were very persistent. Acidity, ulcers of the mouth, red, tender, and coated tongue, were marked features. Disorders of the nervous system, also, were very pronounced. Hysteria, hypochondriasis, headache, impaired memory, and defects of the special senses, are mentioned by Da Costa in the first rank as symptoms. In women, too, the menstruation was deranged, and various diseases of the sexual system were present. In one of the author's cases membranous dysmenorrhœa had existed for some years. As regards the intestinal symptoms, including the passage of pseudo-membrane, variations from the description above given have been noted. The pain may continue during the interval between the paroxysms, although it is much less severe, and the membrane may be present in all the discharges occurring during months or years.

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* "The American Journal of the Medical Sciences," October, 1871, p. 321, *et seq.*

Course, Duration, and Termination.—The course of membranous enteritis is irregular, and the duration indefinite. It may occur in paroxysms of a very acute character in quick succession, lasting two or three weeks or more, and followed by an interval of comparative health, to be succeeded after months or years by the same succession of symptoms. Or the cases may be less acute, and continue for months or even years.

Diagnosis.—The distinction is to be made between membranous enteritis, dysentery, and tape-worm. The passage of shreds and casts of false membrane separates this malady from dysentery, unless there occurs separation or desquamation of the epithelium in the latter, when the aid of the microscope must be invoked. The smallest shreds of false membrane may be confounded with the strobila of a tape-worm colony, but, as the latter has a perfectly well-defined structure, and has the power of independent movement for a short time, only ignorance could possibly hesitate.

Treatment.—The suffering which attends this malady requires relief, and the preparations of opium must be used. The most effective anodyne treatment is the hypodermatic injection of morphine. Next to this are enemata of starch and laudanum. No specific treatment has been proposed, and only symptoms are to be prescribed for. In the author's experience, minute doses of corrosive sublimate, of copper sulphate, and of arsenic persistently used, are the most effective remedies for the more chronic cases; for the acute, an emulsion of almond-oil, or castor-oil and turpentine when there is constipation. The author has had good results from tincture of nux vomica and tincture of physostigma, fifteen to twenty drops of each *ter in die*, for the subacute and chronic cases, and the persistent use of hydrastis, eucalyptus, cubeb (fluid extracts), and other remedies acting similarly, is strongly advised.

DYSENTERY.

Definition.—In common language dysentery is known as "flux"; sometimes as "bloody flux"; in technical, as ulcerative colitis. It is a disease characterized by tormina, tenesmus, mucus, and mucus-and-blood stools, burning pain, with more or less constitutional disturbance. It occurs in the sporadic, endemic, or epidemic form, and in the latter seems to be propagated by a specific virus.

Causes.—It occurs in both sexes and at all ages. Sudden arrest of perspiration by exposure to cold, and especially to cold and dampness combined, is one of the most common causes. Climatic influences are very important factors in its production. It is a disease of those parts of the year in which the change of temperature from night to day is greatest, as in the later summer and autumn, and in warm rather than in cold climates. It is especially prevalent in malarious regions, doubtless because of the congestion of the portal circulation induced by paroxysms of ague. Agents, whether of food or medicine, producing irritation of the mucous membrane, may cause a dysenteric attack. Is there a specific virus? Although during an epidemic the mode of propagation would indicate the existence of a specific infective material—a microbe—it is probable that this is nothing more than the dysenteric discharges themselves acquiring increased virulence by the aggregation of numbers of sick under unfavorable hygienic conditions. The dysenteric excreta undergo certain fermentative changes, probably, by which their infective property receives additional strength. They are admitted to the ground-water in the dried state; finely divided they are distributed by the air, and in many ways, by the atmosphere, food, and drink, they reach the intestinal canal of man, and there induce the characteristic disturbances and structural alterations of dysentery. On the other hand, the virus or infective material of epidemic dysentery may be a ptomaine, produced in great quantity when the *materies morbi* reaches the intestinal canal. As an epidemic, dysentery is a prevalent disease in armies, in jails, in tenement-houses—wherever, indeed, numbers of human beings are crowded together under unfavorable hygienic conditions. Indeed, it seems almost certain that ileo-colitis and ulcerative colitis may be induced by the emanations from fecal accumulations and by the gaseous products of animal decomposition. Unlike contagious and other infective diseases, one attack of dysentery does not confer immunity; in fact, the tendency is increased with the number of attacks.

Pathological Anatomy.—The structural alterations of dysentery may be comprehended in two groups, catarrhal or sero-purulent, and croupous or fibrinous.

The first step in the series of changes occurring in the catarrhal form is an intense hyperæmia, the mucous membrane being of a deep reddish color, with here and there blackish points. The redness is not universal, but at the summits of the mucous folds. This congestion is not limited to the mucous, but extends also to the submucous connective tissue. As a result of this congestion there is over-production of mucus, which is found adherent, but not closely, to the membrane, and the follicles enlarge from an accumulation of their contents, while just around them is a girdle of enlarged vessels. The submucous tissue thickens greatly, and is infiltrated with serum, and this infiltration extends to the muscular layer. Softening of the mucous membrane now ensues; it undergoes disintegration and gradual detachment, leaving still adherent here and there portions of membrane with ragged edges, and a coating of fibrinous pellicle, still in place. The follicles resist the destruction from softening longer than other portions of the membrane, but finally they slough out. The disintegration of the mucous membrane is the result of an enormous multiplication of pus-cells within the interstices; the pressure is increased by the swollen vessels, and rapid necrosis (softening) ensues. Recovery readily takes place in the cases of catarrhal inflammation before the softening begins, and after softening if the destruction is not extensive. Repair is effected by cicatrices, which are much smoother, and, of course, devoid of gland-structures, and are therefore easily recognized. In the fibrinous or diphtheritic dysentery the alterations of structure are very different. The initial change, as in the catarrhal form, is an extensive hyperæmia, but, instead of being confined to the summits of the folds, (valvulæ conniventes of the small intestines, and the folds from contraction of the muscular layer in the large) there is a universal deep, bluish-red congestion of the lower end of the ilium, and the whole of the colon. Extensive extravasations of blood infiltrate the whole tissue of the mucous membrane, but it is especially invaded and transformed by a fibrinous exudation. The proper structure of the mucous membrane disappears entirely, except remains of the tubular glands, and it presents internally a reddish-white surface, variegated with irregular blackish and reddish figures. The result of these changes is to convert the membrane into a dense, parchment-like, and rather unyielding tissue, composed largely of the deposited fibrin. If death do not take place when the alterations of the mucous membrane have reached this point, gangrene ensues. Although the ultimate changes in the two forms of dysentery are so distinct, yet in most cases the alterations found *post mortem* are made up of both forms, the catarrhal and fibrinous. Those parts of the intestinal wall affected by the fibrinous inflammation are thicker and more prominent than those attacked by the catarrhal. Hence the surface is uneven, the fibrinous parts dark from the presence of extravasated blood, or reddish-white where the fibrin predominates. Local gangrene patches appear, in size from a copper cent to a silver dollar; the membrane disintegrates and is detached in considerable sloughs, leaving deep excavations which extend deeper by succeeding necrosis to the peritoneum. The purulent infiltration in those parts, the seat of catarrhal inflammation, also leads to extensive destruction of the submucous layer and large excavations beneath the mucous membrane, which is either detached as a whole, or in turn yields to necrosis. These more superficial catarrhal excavations contrast strongly with the dark-red or blackish sloughs of the fibrinous.

The extent to which the intestine is involved varies greatly. The rectum, the cæcum, or the sigmoid flexure, may be alone involved; the whole of the large intestine, the disease beginning below and extending upward, may be invaded. Repair is possible only when a small extent of the mucous membrane has been destroyed by gangrene. When the morbid process is arrested, the sloughs separate, granulations spring up, and the excavations are closed by cicatrices, which by subsequent contraction may seriously encroach on the lumen of the bowel. The structural alterations are not limited to the mucous, submucous and muscular layers. When the ulcers reach the peritoneum, this membrane becomes cloudy, then intensely injected, and fibrinous exudation forms and adhesions are contracted to neighboring surfaces. When perforation ensues, a limiting inflammation may cut off the injured parts from the general cavity, and form a purulent collection, or general peritonitis may ensue if the shock does not terminate the history of the case.

The mesenteric glands are enlarged, hyperæmic, and softened, and often are broken down into abscesses. The liver is very commonly the seat of numerous small abscesses, from embolic obstruction of the radicles of the portal vein. The lungs present in their dependent parts the changes of hypostasis. The heart is small, flabby, and its muscular tissue more or less fatty.

Symptoms.—In the epidemic form, dysentery may begin suddenly, without any preliminary symptoms, and with great violence, but in the endemic and sporadic form, and in the milder cases during epidemics, there is usually a prodromic or preliminary stage. There is more or less catarrh of the intestines, diarrhœa, chilliness followed by feverishness, toward evening especially, and that state of general discomfort known as *malaise général.*

In the mildest cases of dysentery there is no fever, but when the symptoms are at all pronounced there is fever of a remittent type, the exacerbation occurring toward evening. The type of the fever is, of course, determined by the extent of the local lesions.

When actual dysenteric symptoms come on, which happens in two or three days after the first of the prodromic period, very decided abdominal pain is felt along the course of the descending colon and about the sigmoid flexure, and is increased by pressure at these points. These abdominal pains, felt also somewhat about the umbilicus, are described by the term *tormina*—"colicky pains." There is pain of a burning character in the rectum, but especially a sense of the presence of a foreign body, with the desire to strain for its expulsion. The patient resorts again and again to the close-stool, and makes strong efforts at expulsion, but instead of any fæces being discharged he only brings away some jelly-like matter—mucus—either alone or tinged with blood, and occasionally a hard ball of fæces (*scybala*), but without any relief. The feeling of bearing down (*tenesmus*) and the burning pain felt in the rectum and through the hips continue as before, so that he finds it impossible to quit the stool, or returns every few minutes, and each time he sinks back to bed exhausted and unrelieved. At the beginning, before the characteristic dysenteric stools appear, there are loose fecal evacuations containing mucus, voided with great pain. Presently, however, fæces are no longer present in the evacuations; they consist of a grayish, tough, transparent mucus in pellets or small masses, containing here and there whitish granules, which have been likened to grains of sago. On the second or third day, blood appears in the stools, and the *débris* of epithelium are mixed with the mucus. In the mildest cases, the course of the disease is ended with these manifestations. These do not differ from the mildest cases seen during the existence of an epidemic; on the other hand, the most formidable, the fulminant cases, may occur sporadically. In the more pronounced examples, after three or four days, severer symptoms make their appearance—the amount of blood discharged increases; not only the *débris* of epithelium, but the pellicular neo-membrane (an exudation) and necrosed parts of the mucous membrane are now to be detected in the stools. The stools have no longer any fecal odor, but are very fetid from the presence of gangrenous portions of mucous membrane. The grayish, transparent mucus gives place to a puriform fluid, and there is not only considerable admixture of blood, but a good many clots of pure blood are also discharged, and indeed a real hæmorrhage may occur. A stool may consist of a bloody, purulent fluid and scybala, and the next be composed largely of an extremely fetid, brownish fluid containing bits of neo-membrane and masses, often of considerable size, of decomposing gangrenous sloughs of the mucous membrane. Sometimes a cast of a part of the bowel, consisting of the mucous membrane in a complete cylinder, all of its parts distinct enough for recognition, will be discharged. These ought not to be confounded with the infinitely rarer accident of a slough of the bowel itself, several feet in length, cast off by intussusception. As has already been pointed out, in the catarrhal form of dysentery, deep-seated suppuration in the submucous layer sometimes extends widely, and the mucous membrane sloughs off before it has had time to become gangrenous. During the tormina nausea is often felt, and vomiting occasionally occurs. In the severe cases, vomiting is constantly present and adds materially to the gravity. The vomited matters consist of articles of food and drink, of gastric mucus, and ultimately of biliary matters from the gall-bladder. The bladder in severe cases is also affected by tenesmus. The urine is scanty, high-colored, and very acid, and therefore irritating, and so sensitive does the bladder become that a few drops of urine present in it excite the tenesmus, and in the straining both the bladder and the rectum are simultaneously affected. The frequency of the stools represents pretty nearly the gravity of the case. In the mild cases there may be ten to twenty daily ; in the severe cases forty or fifty, and in the fulminant they may reach a hundred or more. Lessened frequency is a good indication when the character is improved. The amount discharged is small unless hæmorrhage occurs. Artificial distinctions based on the character of the stools have been made, but these have no practical importance. It must be obvious that a disease affecting so large a part of the intestinal mucous membrane, and of so formidable a character in itself, must quickly impair the bodily forces. Even in the mild cases considerable emaciation occurs and the return to health is slow. In the severe cases, systemic infection results from the products of decomposition and from the gangrene, and they wear the aspect peculiar to this state. The weakness early reaches the point that the patient is unable to leave the bed ; the evacuations pass without his control ; the anus and neighboring parts become excoriated and bed-sores quickly form. The face wears an anxious expression and is pinched ; the skin is dry, harsh, and wrinkled ; the pulse small, quick, and feeble. With the most painstaking care the person and bedding of the patient will be fouled with the discharges and emit a horribly fetid odor. From this condition of depression the case passes into the stage of collapse, when the pulse ceases at the wrist and the heart beats very feebly, an obstinate hiccough comes on, the skin is covered with a cold sweat, the hands and feet become cold and livid ; the face is shrunken, the eyes deeply sunk, the voice husky. In this condition the patient usually betrays a singular apathy, although the mind remains clear until the failure of oxygenation of the blood causes carbonic-acid poisoning and stupor. The state of collapse may not come on in this gradual way, but the patient pass suddenly into it, by reason of perforation of the bowel and the resulting shock followed by peritonitis. Death does not necessarily ensue immediately after the symptoms of collapse have been fully developed. The patient may remain in this low state for several days, now presenting delusive appearances of improvement, now declining. Various complications may arise during the course of dysentery. Thrombosis of the intestinal veins, or a form of phlebitis, or the absorption and deposition of some unknown morbific material, may excite inflammation and abscess of the liver. This is a common accident in tropical regions and in the interior of the American Continent. Hepatic abscess is, however, more frequently due to the milder than the severer forms of dysentery, because of the destruction by gangrene and the rupture of vascular communication, which takes place in the latter. It follows disease of the rectum much more commonly than of the colon or cæcum, because of the greater abundance of large vessels in the latter and the comparative sluggishness of the blood-current. Besides abscess of the liver, purulent collections are sometimes found, as the author has seen, in the lymphatics at the root of the lungs and elsewhere. Peritonitis is a usual complication, not due necessarily to perforation, but to the extension of the ulceration to the peritoneum. Increased tenderness of the abdomen and an exacerbation of the systemic symptoms are evidences of the onset of this complication.

Course, Duration, and Termination.—In the mild cases the disease usually begins with diarrhœa; tormina and tenesmus are felt about the second day, when also mucus appears mixed with fæces. About the third day the more characteristic stools are seen, and the disease has attained its height on the fifth and sixth days, when improvement begins, and convalescence is established about the eighth day. The signs of improvement are, a diminution in the number and frequency of the stools, the reappearance of fæces, and the disappearance first of the blood and next of the mucus. In the more severe cases the duration is more protracted. The maximum in the intensity of the symptoms continues for several days; the state of adynamia is more serious and prolonged, and the return toward health may be by almost insensible gradations, lasting several days. The prodromic period in such cases will be about three days, the fully developed period will range from four days to a week, and the period of gradual improvement will last about the same time, so that the whole duration of such a case will be about three weeks, while the convalescence will require a month for full restoration to health. The termination may be in partial recovery, or in chronic dysentery. When this is the case, the more severe symptoms subside, the stools improve in character, but they never become entirely healthy, and the general condition is more favorable. Now fecal stools, with only a little mucus and blood, are passed, but these may be succeeded by evacuations entirely of pus and blood. With this varying fortune the case may proceed for months, even years, the patient in a feeble state, emaciated, and yet able to keep out of bed, or so reduced as to be unable to sit up except for a little while every day. The prolonged suppuration in these cases induces amyloid degeneration of the liver, spleen, and kidneys, the ultimate result being albuminuria and anasarca.

Another mode of partial recovery is narrowing, contraction, and deformation of the bowels, the effect of which is to impair assimilation and nutrition, so that after a period of improvement a progressive loss of flesh and strength is observed, and ultimately death occurs by exhaustion.

Prognosis.—Opinions must be expressed with caution in the early stages of dysentery, for it is not then possible to estimate correctly the extent of the inflammation, nor its form. A favorable prognosis can be given in those cases which continue mild, and even in severe cases, if the signs of collapse are absent. Whenever the symptoms begin with great violence (fulminant form) a guarded prognosis is judicious. If the symptoms of collapse are persistent, especially if gangrenous sloughs appear in the stools, an unfavorable opinion must be given. In severe and protracted cases that are apparently improving, the probability of a partial recovery should not be lost sight of.

Diagnosis.—The symptoms are so characteristic that a differentiation is rarely required, except as between simple and acute catarrh of the rectum (proctitis) and dysentery proper. The dysenteric symptoms in proctitis are much less severe; the discharges consist of mucus and muco-pus, sometimes intermixed with blood, but never the foul discharges of dysentery, the shreds of false membrane, the gangrenous sloughs, etc., which constitute so characteristic an evacuation. In croupous enteritis, which is as rare as dysentery is common, there are discharges of shreds of pseudo-membrane with tormina and tenesmus, but the attacks are paroxysmal, the evacuations continue the same, and the subsequent history is widely different from that of dysentery.

Treatment.—As in this disease the nutrition of the body suffers severely, the right use of aliment is important from the beginning. If the stomach is irritable, milk, with one fourth lime-water, is the best food. If there is but little nausea, and especially if the digestion remains good, the patient can take milk, eggs, beef-juice, ice-cream, boiled custard, oyster-soups, mutton, chicken, and beef broth, and similar articles, but solids and aliments generally leaving much residuum, and especially coarse articles, are highly objectionable, because they increase by friction the irritation of the inflamed membrane. Where there is much depression of the powers of life, egg-nogg (milk, egg, and brandy) may be freely given, and champagne be used to allay vomiting.

Of medicinal measures, the treatment by saline laxatives is of the highest importance. Bretonneau, preceptor, and Trousseau, pupil, strongly urged the sulphates, and the author is convinced that the sulphate of magnesia in solution with dilute sulphuric acid is entitled to the first place as a remedy. It must be given in laxative doses, and at the right time—that is, before the mucous membrane has begun the process of disintegration. It serves a triple purpose: it empties the canal of retained fæces; it lessens hyperæmia by setting up an outward diffusion; its after-effect is astringent and sedative. Next to the sulphate of magnesia, and by many given the first place, is ipecac. The experience with this remedy, ancient and modern, is now so great that the limit of its curative power is well and accurately defined. It is applicable to the first stage of dysentery, before the mucous membrane is stripped off. It must be given, according to recent Indian experiences, in which the author in the main concurs, in scruple to drachm doses, every four to six hours. The effects to be derived from it are these: The first doses empty the stomach thoroughly, then a tolerance is established, and the considerable doses prescribed are carried quietly by the stomach, but act on the intestinal canal, producing copious bilious evacuations, so characteristic as to be called "ipecac-stools"; after the purgative action ceases a calmative and astringent action continues. The utility of ipecacuanha ceases with the production of the characteristic stools, and very decided amelioration in the remediable cases usually follows. There is one form of dysentery, above all others, in which the ipecac-treatment is signally beneficial—the puerperal. The author has witnessed some remarkable cures in cases of puerperal dysentery, a disease which is well known to be very dangerous to life. As regards the dose, the large quantity of a drachm prescribed by our Indian colleagues seems unnecessary in our temperate climate. It will usually be necessary to give twenty grains at a dose. It is best administered in milk, and is better borne if some aromatic powder is added to it. The next remedy in point of efficiency for the treatment of the first stage of dysentery is castor-oil, administered in purgative doses, for the purpose of ridding the canal of acrid and fermenting materials, and of retained fæces, and to secure the after-quietude which succeeds to the action of a purgative. After using one of the agents of the cathartic group as above directed, what remedies are most appropriate for the treatment of that condition in which either purulent or fibrinous infiltration, or both, is taking place? Under these circumstances an emulsion of oil (almond-oil) and turpentine is very serviceable, and combined with opium, if the pain be very severe. When destruction of the mucous membrane is beginning, the most effective remedies are corrosive sublimate, sulphate of copper, sulphate and oxide of zinc, acetate of lead, bismuth, arsenic, etc. Of this formidable list, sulphate of copper and arsenic are most effective. They ought to be combined with opium. The author has had excellent results from the use of Fowler's solution, one drop, and deodorized tincture of opium, five to twenty drops every three hours. Sulphate of copper must be given in small doses (one twentieth of a grain) every three hours, with morphine (one eighth to one twelfth of a grain). Bismuth in large dose (℈ j—℈ ij) every four hours is sometimes beneficial, especially if administered with carbolic acid. When there is much fetor and sloughs are threatening, good effects are obtained from naphthaline (two grains every three or four hours), carbolic acid, thymol, etc. Numerous vegetable astringents, owing their therapeutical power to the tannic acid which they contain, have been much employed, with more or less advantage, but they are not equal to the mineral astringents. Applications to the rectum and colon are unquestionably useful. By the method of irrigation the whole of the colon may be safely reached. Excellent results are obtained by washing out the bowels with warm water (100° to 105° Fahr.). The patient is placed on his right side, the thighs well flexed on the pelvis, the hips elevated and brought to the margin of the bed, the chest and head on a lower level. The anal tube is inserted two or three inches, and the reservoir is placed at a sufficient height to insure the passage of the water. Various demulcent applications may also be made in this way. Very great relief is afforded by the injections of starch and laudanum after an evacuation, or especially after irrigation and washing out the bowels. Much emphasis should be put on the employment of nitrate of silver enemata. They possess a high degree of utility if efficiently administered. A tube which is not acted on by the silver salt should be passed carefully up to the sigmoid flexure, and about eight ounces of a strong solution of silver nitrate (℈j—ʒj to the ounce) should be thrown up. The time for performing this is after sufficient quiet has been obtained by the hypodermatic injection of morphine. So rapidly is the insoluble chloride of silver formed that no ill results can follow the strongest solution employed for this purpose ; but, if there be any reason to apprehend mischief, a solution of common salt may be injected immediately after the silver.

If the injections are, for any reason, inadmissible, suppositories of cacao-butter containing morphine, morphine and tannin, morphine, or opium and acetate of lead, etc., can be used instead. Lately injections and suppositories of fluid extract of ergot, and of ergotin, have been used, and apparently with good results. Ergotin has been given internally, and, in some epidemics, with an apparent utility which the physiological effects probably warrant. It is difficult to understand how it can accomplish anything when in the catarrhal inflammation the mucous membrane is infiltrated with pus, and in the croupous with fibrin. After the use of the saline laxative, or the ipecac, the morbid process continuing, is there no means of securing that quietude of the intestine which will permit the mineral astringent to act on the diseased surface? The author believes that we possess such an agent in the hypodermatic injection of morphine. He therefore urges, from the point of view of personal experience, this means of treatment. Besides giving the remedies an opportunity to act on the diseased surface, the morphine injections suspend that violent reflex peristalsis which does so much injury to the diseased mucous membrane. External applications, if not curative, are grateful. The cold wet pack, the ice-bag, and other cold applications, are sometimes preferred ; but generally warm —rather hot—applications afford more relief. The turpentine stupe is generally more useful than other warm applications. With the beginning of the symptoms of collapse, active stimulation may be necessary. The best form of stimulant is cognac brandy, as it is at the same time astringent. Beef-juice and brandy, milk and brandy, and egg-nogg, are combinations of food and stimulant most generally useful. As already indicated, the strength must be supported from the outset by suitable nutriment. It is necessary to keep the person of the patient and the bedclothing clean. The discharge should be removed from the apartment as soon as passed, and should be thoroughly disinfected before going into the common receptacle. A strong solution of sulphate of iron is a cheap and effective agent for this purpose. Some tincture of iodine exposed in a saucer is an excellent deodorizer for the apartment of the patient.

ULCERS OF THE INTESTINES.

Forms.—Ulcers of the intestinal canal exist in three forms :

Ulcers from mechanical irritation.

Ulcers from thrombosis or embolism.

Ulcers from tuberculous deposit.

There are duodenal ulcers, cæcal ulcers, and rectal ulcers, and the chief cause is cirrhosis of the liver ; hence it is convenient, in the description, to study these ulcers, according to their anatomical position, going from above downward.

The Nature, Symptoms, and Treatment of Ulcers of the Duodenum.—The first or transverse part of the duodenum is the almost exclusive seat of the ulcer. The pathological history of this ulcer is the same as the corresponding ulcer of the stomach. The great factor in its causation is thrombosis, or embolic obstruction of a vessel. An admirable instance of this accident (the embolus in position, the ulcer forming) has been reported,* confirming clinically that which had previously been demonstrated by pathological experimentation. When the blood-supply has been cut off from a part of the mucous membrane, the digestive juice, no longer opposed by the alkaline stratum beneath, dissolves or digests the membrane, and an ulcer is formed. At first it is a round, smooth, sharply defined ulcer, but the inflammation which is lighted up cuts off the action of the gastric juice from the adjacent healthy tissues, by a deposit of new material of a granulation-tissue structure, and especially protects the bottom of the excavation ; otherwise perforation would quickly ensue in most cases. As the layers of the duodenum are invaded, not all at once, but successively, and as the distribution of the vessels is fan-shaped, it is obvious that the resulting ulcer must have shelving margins and a stratified appearance. The term " crater-like " aptly enough describes its characteristics.

This description of the process by which duodenal ulcers are formed can be applicable to ulcers situated in the first part of the duodenum only, for, soon after the acid contents of the stomach reach the vertical part, they begin to have an alkaline reaction. It is in the first part that the ulcers are found, and they are sometimes partly in the stomach and partly in the duodenum. They are usually single, and occasionally multiple. The cause that gives origin to one may produce several (emboli), so that it is not uncommon to find gastric and duodenal ulcers existing at the same time. As regards the relative frequency in the occurrence of ulcers in the stomach and duodenum, respectively, they are found in the former organ thirty times more frequently than in the latter. The duodenal ulcer is found between thirty and forty years of age in a great majority of cases, and becomes very rare after sixty (Krauss).* As to sex, the preponderance is in favor of males, and is so extraordinary in proportion as fifty-eight to six. Accident in the collection of cases had something to do with these figures. Besides the causes already mentioned, burns of the skin, especially of the chest and abdomen, have induced ulceration of the duodenum. The burns must be of considerable extent to bring it about, sufficient to cause a reflex spasm of the vessels, thus permitting the gastric juice to act on the membrane. If the ulceration reaches the peritoneum adhesions may be contracted to neighboring organs, to the stomach, pancreas, gall-bladder, etc., and fistulous communications may be established ultimately between them. In the process of widening of the ulcer, a vessel may be opened and hæmorrhage result, a very common symptom, occurring in one half of the cases. By perforation a local peritonitis may be set up, adhesions contracted, and a cavity containing sero-purulent fluid, shreds of tissue, etc., formed; or the general cavity of the peritoneum may be entered and general peritonitis excited. When an ulcer of the duodenum heals, the puckered cicatrix which results may induce remarkable changes. Contraction of the pyloric orifice and dilatation of the stomach will be results of the cicatrization of an ulcer situated at the entrance to the duodenum; if lower down, the lumen of the bowel will be encroached on, and dilatation occur above the contraction. An ulcer may be so situated that the pancreatic and common duct of the liver will be obstructed with the usual results of such obstruction. Ulcers of the duodenum situated near the pyloric orifice will be accompanied by some of the symptoms of a gastric ulcer situated at or near the pylorus. Vomiting is a pretty nearly constant symptom, coming on several hours after eating. Tenderness to pressure, and, when the ulceration approaches the peritoneal surface, rather exquisite tenderness, is felt in the position of the duodenum. Attacks of gastralgia, of enteralgia rather, and of a severe character, occur under the same circumstances as gastralgia in stomach-ulcer. The pain is distributed through the solar plexus and the hepatic plexus also, and is of a very depressing kind, the action of the heart becoming exceedingly feeble, the surface cold, etc. Jaundice may also be present. When this is the case, it would be impossible to differentiate between ulcer of the duodenum and hepatic colic. Hæmorrhage may take place by emesis or by stool. In duodenal ulcer it may, in consequence of the size of the vessel (the ascending vena cava, for example), be so large as to cause death immediately. The blood, unless in large amount, is much changed in character by the action of the intestinal juices, as has been pointed out. The diagnosis may be aided by a study of the hæmorrhage, the part discharged by vomit having the characteristics of hæmatemesis, that passed by stool presenting the appropriate changes. As regards treatment of ulcer of the duodenum, the plan proposed for gastric ulcer is applicable. (See ULCER OF THE STOMACH.) Ulcers similar in character to the duodenal, but due to those alterations of the vessels which occur in amyloid degeneration, are occasionally found in other parts of the small intestines. The symptoms are obscure, and the diagnosis a mere matter of suspicion. The patient affected with an ulcer of this kind suffers with the changes wrought by amyloid degeneration, in the liver, kidney, spleen, and other organs. There are emaciation, pallor, œdema, diarrhœa, etc., and there may be soreness in a particular locality, and hemorrhage, to indicate the nature of the intestinal disease, but obviously these are far from conclusive. The general condition is the point to which attention must be directed in these cases, yet no subject in therapeutics is more unsatisfactory than the amyloid disease.

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* Merkel, "Wiener Presse," various numbers in 1866.

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* "Das perforirende Geschwür im Duodenum," Berlin, 1865, p. 24.

The Nature, Symptoms, and Treatment of Ulcers of the Cæcum and Appendix Vermiformis.—Ulcers in these situations are usually of mechanical origin, produced by the retention of hardened fæces, by the impaction of an intestinal or biliary calculus, or of another foreign body, such as a grape-seed, a cherry-seed, a pin, etc. These foreign bodies lodge more frequently in the appendix vermiformis, but they may become impacted in a fold of the mucous membrane of the cæcum, especially of the posterior wall, for this has a fixed position. The pressure of the foreign body excites inflammation, then softening, and finally perforation. The position of the ulcer affects the result enormously. If it perforate the posterior wall of the cæcum, which is not covered by the peritoneum, the foreign body and other contents of the bowel escape into the loose connective tissue, where an inflammation ending in an abscess is set up. Then the history is that of fecal abscess. Occasionally a primary inflammation develops in the pericæcal connective tissue, an abscess forms, and a communication is established with the bowel. The author has had the opportunity to study a case of this kind which lasted two years, and at the autopsy a large pus-cavity in the iliac fossa behind the cæcum communicated with the cæcum by a considerable orifice. As the discharges of matter through the bowel had been paroxysmal, it is probable that the original opening was small.

If the foreign body is lodged in the appendix, inflammation is excited, and a perforating ulcer quickly formed. In some cases the whole appendix is inflamed and converted into a diffluent mass. As the ulcer extends, the peritoneum is quickly reached. One of two results must then take place: either a local peritonitis with adhesions, limiting the mischief to that locality, or a sudden rupture into the general cavity of the peritoneum. If the process is slow, the peritoneum forms adhesions to the neighboring surfaces; if rapid, the time is not sufficient to accomplish this. When a limiting inflammation is thus developed, a cavity is formed, containing the matters which have escaped from the appendix, including any foreign body lodged there, fecal matters, sloughs of the ulcerated surface, serum, and pus. In a short time the process of extrusion begins, the pus makes its way downward under Poupart's ligament, along the sheath of the femoral vessels, and points in the usual situation. In two thirds of the cases the purulent collection takes this direction; in others it points over the crest of the ilium, and posteriorly, in the lumbar region. Besides the ulcers of merely mechanical origin, the cæcum is the seat of that form of ulcer known as the catarrhal—a fact which the author believes he was the first to demonstrate.* It is a fortunate circumstance that these catarrhal ulcers, which have such a strong tendency to perforate the bowel, are usually situated on the posterior wall; doubtless in accordance with the now well-known law that those parts most exposed to injury in the performance of their functions are also most liable to disease. As may be seen by referring to the article on "Typhlitis," the symptomatology and treatment are the same as for ulcer, and indeed there is no well-marked distinction between them clinically, except it may be the vague symptoms of ulcer which precede the perforation for an indefinite period. The rectum is also the seat of ulceration of the catarrhal type. This has already been pointed out, and its symptomatology demonstrated, but more frequently ulcers of the rectum have a mechanical origin, are brought on by impacted fæces, the lodgment of a fish or other bone, of seeds, etc. Perforation ensues, an abscess is formed, which points alongside the rectum, in the perinæum and elsewhere, leaving troublesome fistulæ. An ulcer of the rectum, healing, may produce narrowing and deformity of the bowel, seriously impairing its functions. But these ulcers of the rectum do not heal readily, for obvious reasons—the frequent muscular movements, the passage of rough matters over them, the constant presence of irritating solids, fluids, and gases, etc.

As regards the treatment of ulcer of the rectum, there are two points—to keep the bowels soluble without frequent motions, and to make topical applications of the solid nitrate of silver. To this might be added a third—stretching the sphincter. This can be done by a bivalve rectal speculum, working with a screw, when the parts are exposed for the applications to the surface of the ulcer.

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* "On Typhlitis and Perityphlitis," "Amer. Jour. of Med. Sci.," October, 1866, p. 351.

The Nature, Symptoms, and Treatment of Tuberculous Ulcers.—Ulcers of tubercular origin are not limited to any anatomical division of the intestine, but they occur most frequently in the lower end of the ileum, to which, indeed, they may be entirely confined. They may occupy the whole extent of the mucous membrane from the stomach to the rectum; they may be confined to the cæcum, appendix, and colon.

The deposit of miliary tubercle takes place in the follicles, which become crowded and obstructed, so that the cells undergo fatty degeneration and atrophy. The miliary tubercle, in preparation for extrusion, becomes caseous, softens, and carries with it the surrounding textures, thus forming an ulcer, which widens by the addition of new miliary tubercle, destined to undergo the same process of caseation, softening, and extrusion. The situation of the ulcers has reference chiefly to the distribution of the vessels, which is transversely, and on this anatomical fact has been based a means of distinguishing between tubercular and catarrhal ulcers. This is true only of the early stage of the tubercle deposit, and can no longer be depended on when, as subsequently happens, the formation of the ulcers takes place longitudinally also. By coalescence their form is greatly altered. The extension of tubercle-ulcers through the muscular layer of the bowel is very slow, and takes place chiefly along the lymphatics, ultimately reaching the peritoneum. Indeed, it is easy to trace with the naked eye the tubercle-masses crowding the lymph-vessels and the lymph-spaces adjacent. Deposits then cloud the peritoneum, a patchy exudation forms, and adhesions connect the neighboring serous surfaces, and so usual is this result that perforation by a tubercle-ulcer is rather uncommon. Tuberculosis of the intestinal mucous membrane is a local manifestation of a general state; hence, when these ulcers exist in the intestines, tubercular deposits will be found elsewhere. The most characteristic symptom of tubercular ulcerations is an obstinate diarrhœa, which resists every means of treatment, and is only palliated. The stools are usually yellowish, are very thin, and contain pus, small sloughs of the mucous membrane, etc., and are very fetid in odor. Colicky pains attend them, and tenesmus also, when, as is frequently the case, the rectum is involved. The stools contain also small, whitish lumps (sago-grains), masses of mucus extruded from those spaces which had contained the follicles. Clots of blood, an admixture of pus and blood, and of liquid fæces and blood, are also contained in the evacuations. The approach of the ulcers to the peritoneal surface is recognized by the increased pain, and the tenderness to pressure at various points. The general condition of the patient is highly significant. Emaciation proceeds rapidly. The evening temperature is high (103°

-105° Fahr.), and the fever is distinctly septicæmic in type. There is, at the same time, pulmonary mischief going on, as a rule, in these cases. Investigation will disclose the fact that an hereditary tendency exists. The treatment consists in the use of opium and astringents, vegetable and mineral. In the course of treatment of an ordinary case, all the resources of the materia medica in remedies of this kind will be exhausted. Under the heading of "Intestinal Catarrh" will be found some remarks on treatment equally applicable in this malady.

CANCER OF THE INTESTINES.

Forms and Site.—The three forms—scirrhus, medullary, and colloid—which affect the stomach, occur also in the intestines. As has been stated already in regard to cancer of the stomach, the origin of the neoplasm is epithelial, and the initial change (always, however, preceded by a pronounced local hyperæmia) is a proliferation of the cells of the follicles. The new cells extend downward and develop in greatest abundance in the submucous layer. The growth takes an annular direction, and in the contraction, which always results, the lumen of the bowel is encroached on and stenosis produced. As a rule, those parts of the bowel most active functionally, and in a situation to be most readily injured in the performance of their functions, are most apt to be the seat of cancer; the rectum, the cæcum, and the flexures of the colon, are these parts.

Cancer of the intestine is usually primary. It is a disease of advanced life (after forty), although the soft variety, the medullary, may occur at any age.

Symptoms.—There are three symptoms which have a high degree of significance: pain in a fixed situation; a gradually developing cachexia; the presence of a tumor. Until these symptoms appear, the diagnosis will be largely conjectural. The pain is at first a mere vague uneasiness; gradually a sensation of soreness with some tenderness to pressure is developed, and finally there are two kinds of pain—a dull, heavy, tensive soreness, and acute, sharp, lightning-like pains. The pain may radiate somewhat from a center, but the most important characteristic of the cancer-pain is its fixed position. From the moment pain is felt in a part the patient declines in strength and weight, and experiences a feeling of fatigue quite irrespective of any exertion. The complexion slowly changes, until ultimately the fawn-color becomes well marked. The lips are then bluish white, the surface dry and scurfy, the skin wrinkled, the hair dry and dead-like. In cancer of the stomach and intestines the patients usually suffer from a profuse salivary flow without apparent cause. Sometimes just above the clavicle may be felt enlarged lymphatic glands. When the emaciation has removed the fat from the abdomen, a tumor can be felt. Although cancer may form anywhere, it is at certain points where we may expect to detect a tumor—the points of election already mentioned. In six cases of cancer of the intestinal canal, observed by the author with special reference to this account of the disease, there were two of the rectum, two of the cæcum, one at the sigmoid flexure, and one at the angle of the transverse and descending colon. If the tumor is scirrhus, it is felt as a hard, nodular mass ; if encephaloid, an irregular growth, partly hard and partly elastic ; if colloid, a more diffused, less irregular and softer mass, not well defined. Very great mistakes are made as to the size of a tumor, or indeed as to its presence, in cases of cancer. As the stenosis increases, accumulations take place behind the point of narrowing, and then hard lumps of fæces may easily be confounded with a nodular tumor. Subsequently the passage of the fæces will give a very different impression, and the real tumor may be detected with difficulty or not at all. The author has observed this state of things in cancer of the cæcum and of the flexures. The symptomatology of intestinal cancer varies with the site of the neoplasm. When situated at the cæcum, pain is felt in the right iliac fossa ; there the tumor may be detected, and there the patient experiences the sensations due to the passage of gas and fæces through a narrowed orifice. Large accumulations of lumps of fæces and gas may occur at times, presenting the appearance of a large tumor, and may disappear spontaneously in a day or two, or be made to disappear by gentle pressure and friction, when they pass through the orifice with a sensation of burning pain to the patient and with gurgling quite audible to those around. The same phenomena occur at the flexures when cancer is developing. In the rectum there is severe, burning pain, of a most agonizing kind, whenever the bowels are moved, or indeed in sitting or standing long, and pains radiate through the hips, thighs, and testes. Usually tenesmus is present, and a constant desire to go to stool, when every attempt at defecation causes unendurable pain, so that the patient, if possible, postpones the painful act as long as he can. The exploration of the rectum by the finger will furnish valuable information : hard nodules will be encountered, and masses may be detached from the ulcerating surface for microscopic examination. In one case the author found protrusion of the rectum and cancer-masses projecting through the anus, while the surrounding tissue (the rectal fossæ) were covered over with enlarged veins and filled with nodes of stony hardness. The least attempt at exploration caused intolerable anguish, and the passage of fæces was accomplished by no less suffering. The stools at first only indicate, if they are solid, that they were forced through a narrowed orifice ; they may be loose or constipated In the progress of the cases, mucus, muco-pus, pus and blood, foul smelling gangrenous masses, and parts of the neoplasm, successively appear and mark the stages in the growth of the cancer. With the increasing stenosis the bowels are less completely emptied; great accumulations finally take place; and, ultimately, death may be brought about by the protracted constipation. When cancer is situated in the first part of the duodenum, it will finally be accompanied by jaundice and the symptoms of gastric cancer at the pylorus, so that it will be impossible to diagnosticate its position correctly—a failure of little moment.

Rupture of the intestine may be caused by an extension of the growth to the peritoneum.

Course, Duration, and Termination.—Cancer goes on steadily to a fatal termination, with now and then some delusive appearances of improvement. The course and duration vary somewhat with age, powers of resistance, and situation of the neoplasm. Cancer of the colon, unless it develops in a way to cause obstruction of the bowel at an early period, is not so quickly fatal as cancer of the cæcum. Cancer of the duodenum interferes so much with digestion and assimilation, and with the hepatic functions, that it causes death by exhaustion comparatively early. A severe hæmorrhage from cancer in any situation may determine a fatal result. The duration varies according to the mode of termination; from one to three years may be regarded as the range. The termination may be by hæmorrhage, by perforation and peritonitis, by exhaustion, or by an intercurrent disease—as pneumonia, pleuritis, pericarditis, etc.

Diagnosis.—When there is no pain, but a feeling of uneasiness, no tumor has formed, no cachexia developed, a diagnosis will be impossible. From catarrh and ulcer of the intestines, cancer is to be differentiated by the age of the subject, the presence of a tumor, and the gradual appearance of a cachexia. The tumor of cancer may be confounded with floating kidney, aneurism, fecal accumulations, and other growths. Floating kidney is a movable tumor, felt in different positions, in which there may be occasional bowel attacks but no persistent disease, and there is no cachexia. Aneurism is a pulsating tumor, with an expansile movement, and the pulsation in one or both femorals is retarded by it and altered in character. An apparent pulsation is imparted to a cancer of the colon by lying over the aorta; but, if moved away by external palpation, or by a change in the position of the patient, the pulsation ceases, and at no time are the femorals affected. A cancer of the cæcum and of the sigmoid flexure may also come into relation to aneurism of the iliac arteries. The same rules apply as above given.

A fecal tumor with colic may cause the merely local symptoms of cancer; but the history of the case, it may be the age of the subject, will decide, and the cachexia will be wanting. The use of purgatives will settle the question.

Prognosis.—No means are now known by which cancer can be arrested in its course, much less cured, so that the prognosis is entirely unfavorable.

Treatment.—Although there are no curative measures to be undertaken, much can be done to alleviate the distresses of the unfortunate subjects. The most easily digested food, and the varieties which can be utilized by the digestive organs without leaving any residuum, should be directed. The bowels should be kept in a soluble state to prevent accumulations, and to avoid friction of the hardened fæces on an irritable surface. To relieve the pain anodynes become necessary, but the physician must carefully guard their administration, owing to the enormous quantity which the patient will use if left to his own inclination. The author must repeat the statement which he has already made in regard to the utility of arsenic in cancer to relieve pain and retard the growth.

INTESTINAL HÆMORRHAGE.

Causes, Symptoms, and Diagnosis.—The subject of gastric hæmorrhage, which has been fully treated, is occupied with the same questions, except the difference in position, as intestinal hæmorrhage; and therefore only a comprehensive but concise statement is necessary here.

Hæmorrhage from the intestines arises from all those morbid states which increase the blood-pressure in the portal system—as obstructive diseases of the heart and great vessels, of the lungs, and of the liver, especially; from rupture of the vessels themselves occurring in the various kinds of ulceration of the mucous membranes, and from morbid states of the blood itself, as purpura, etc. The symptoms produced by an intestinal hæmorrhage will vary with the immediate cause, with the amount of blood lost, and with the condition of the patient at the time. If considerable, the face becomes deadly pale, the eyes glassy; there is a rushing and roaring in the ears; the pulse becomes weak, or ceases at the wrist; consciousness is lost, and a convulsive shudder passes through the muscular system, and death may ensue, without any escape of blood externally: or there may be mere faintness, and consciousness not lost; a sudden and irresistible desire to have an evacuation of the bowels is felt, and blood in clots and partly fluid, or a blackish, semifluid, tarry mixture may be passed. When the hæmorrhage is from the descending colon, the blood discharged—if passed immediately—is unaffected by the intestinal juices, but, if it come from a point high up in the small intestines, it will appear as an homogeneous, tarry fluid, but may, of course, be mixed with fæces. When the blood escapes in small quantity, and slowly, there will not be any systemic evidences of the loss, except a slowly developing anæmia, and the appearance of the blood in the stools will take place in the form already described. When the blood escapes from the rectum it may be passed before, with, or after the fæces, which may be covered with blood, but are not mixed with it. The rectum offers great facility for the determination of the source of the hæmorrhage, and an examination will show whether the bleeding is from hæmorrhoids or from an ulcerated surface. When an ulcer of the rectum exists, the passage of the fæces will cause some blood to flow, which will often be found on the top of the fæces, together with some pus. The importance of intestinal hæmorrhage will depend, first, on the nature of the malady which is its cause; and, second, on the amount of blood lost. If typhoid, or cancer, for example, the importance of the hæmorrhage—unless itself sufficient to cause death—is merged completely in the importance of the malady associated with it.

Treatment.—In the remedial management of intestinal hæmorrhage, the same principles and methods are applicable as were recommended in the cognate disease—gastric hæmorrhage. The most absolute quiet must be maintained, mustard-plasters and ice-bags applied to the abdomen, barium chloride (¼ gr. at a dose), ergotin injected subcutaneously, and alum-whey drunk freely. If time is afforded, the usual iron styptics can be administered by the stomach, or if the source of the hæmorrhage is low down they can be administered more efficiently by the method of irrigation or by enemata. The author having known of an instance of fatal hæmorrhage induced by an injection of a solution of Monsel's salt, given to arrest a hæmorrhage, he advises caution. An intestinal hæmorrhage is a mere symptom; the treatment of it is necessarily a part of the disease with which it is associated. If it occur during the course of typhoid, very different management will be requisite from that necessary in purpura, or in cirrhosis, etc. Only general rules can therefore be indicated here.

ENTERALGIA: NEURALGIA OF THE INTESTINES—COLIC.

Definition.—The term enteralgia is applied to a neuralgia of the intestines, of a functional character, and is therefore a neurosis, and should be studied with the group of *neuroses*, but it is convenient to take it up at this point.

Causes, Symptoms, and Diagnosis.—Except for the difference in site, the story of gastralgia might be repeated here. A more condensed description than would otherwise be proper will now suffice.

The causes of this affection can be comprehended in two groups: an irritable state of the nerves themselves; irritation, by various objects, of the terminal filaments of the nerves (end-organs) in the mucous membrane of the intestinal canal. In the first group must be placed that condition of the nervous system existing in hysteria, hypochondriasis, and in the various cachexiæ—paludal, plumbic, cupric, syphilitic, etc.; and in the second, improper food, coarse and irritant articles, as husks of grain, seeds of fruits, etc.; hardened fæces, impactions of fæces, fermentation and flatulent distention of the bowels; cold, etc.

An attack of colic may come on gradually with a feeling of uneasiness in the bowels, some nausea, eructations of gas, etc., or it may begin abruptly and develop full force at once. When it occurs by either mode, there is felt about the umbilicus a peculiarly severe and depressing pain, having the well-known griping quality. There are numberless gradations in the severity of the attacks, from a little griping pain felt for a few minutes, up to a seizure of such severity that the patient may appear as if collapsed. In any case of moderate severity, the suffering during the time the attack lasts is great—the patient groans or cries with anguish, the body is doubled up, and the fists are pressed deeply in the abdomen; or the abdomen is lain upon with the whole weight. Meanwhile the pulse is small and weak, the surface cool or cold, the face has an anxious and suffering expression, and is covered with a cold sweat. The abdomen may be hard and tympanitic or retracted, and occasionally tender, instead of pressure giving relief. The kidneys secrete a large quantity of pale urine, and a frequent desire to micturate is usually felt. Vomiting generally occurs, and affords some relief, but an action of the bowels, which is always sought for, removes all the pain, at least for the time. Sometimes the attack terminates by a discharge of flatus, by eructation or by the bowels, and then relief is experienced.

The duration of the attacks is variable—they last from a half hour to several hours, and a succession of attacks is not unusual, carrying the case on for several days. When the attacks are plumbic, the colic is known as dry, and obstinate constipation is a prominent symptom—the pain continuing until this is removed. The history of the individual, his occupation as a painter, and the behavior of the case itself, will indicate the nature of the attack. When it is paludal (malarious), the attacks will be distinctly periodical. If syphilitic, the pain will occur in the evening, and leave the patient unmolested during the day. The duration of those cases having their origin in a cachexia will depend on the treatment; for, if the underlying morbid cause fail to be recognized, they may be prolonged indefinitely.

Enteralgia may at once be distinguished from all inflammatory affections by the absence of fever, and of tenderness on pressure, and by the early termination of the seizure, leaving the *status in quo*. It is distinguished from gastralgia by the situation of the pain, and by the relief obtained by an escape of flatus and by an evacuation of the bowels, instead of by vomiting. It is distinguished from hepatic colic by the seat of the pain in the latter, by the tenderness over the gall-bladder, by the appearance of bile-pigment in the urine, and afterward of jaundice. It is distinguished from nephritic colic by the following symptoms which indicate the latter: by the pain along the course of the ureter, by the pain in and retraction of the corresponding testicle, by the strangury and bloody urine, etc.

The colic of gaseous accumulation is differentiated from the other forms by the fullness and tympanitic distention of the abdomen, and by the passage of gas in both directions. This is the colic of infants. The colic of fecal accumulation is recognized by the fullness of some particular part, and the occurrence of pain in the same locality, frequently the cæcum and ascending colon, and at the sigmoid flexure. The colic of lead is associated with the lead-cachexia, with pallor and anæmia, with a blue line along the margin of the gum, with a slow pulse, with a retracted abdomen, etc. The enteralgia of chronic malarial poisoning is known by its prompt occurrence at a fixed time, as has been pointed out.

The prognosis is favorable in genuine colic.

Treatment.—The important point is to remove the cause which gives rise to the disturbance—if some irritant matters or fecal accumulation, an active purgative is indicated. The flatulent colic of infants is quickly and safely relieved by the bromide of potassium and oil of anise in an emulsion—five grains of the former and the eighth of a drop of the latter, every half hour until relieved. For the immediate relief there is no remedy comparable to the hypodermatic injection of morphia and atropia. By relaxing spasm, the injection favors the action of laxatives or purgatives. For the treatment of the colic of some cachexiæ, the appropriate remedies for the cachexia will be necessary: for example, quinia in intermittent colic, iodide of potassium in nocturnal colic, and alum in lead-colic. For the hysterical colic, a combination of Hoffman's anodyne and fluid extract of valerian is effective. Enemata of asafœtida mixture may also be used. For chronic enteralgia of the bowel—an extremely obstinate affection—arsenic, probably, stands in the front rank. The neuralgiæ are, however, considered more fully in another place, to which the reader is referred.

CONSTIPATION.

Definition.—As the term *constipation* is usually employed, it signifies a state of the intestinal canal in which the alvine evacuations too seldom occur. Obstruction of the bowels, whether due to impaction, concretions, tumors, or other causes, is treated of in a separate chapter.

Causes.—Omitting from consideration all the conditions inducing obstruction, the causes of constipation are resolved into three groups: 1. Those arising in deficient secretion; 2. Those due to imperfect action of the muscular layer of the bowel; 3. Those dependent on derangement of the nervous apparatus. When from any cause the intestinal juices, the secretions of the liver and pancreas, are reduced below the normal, a necessary stimulus to intestinal action is removed. As the peristaltic movements are derived from the contractility of the circular and longitudinal fibres of the bowel, it follows that, when their power of contraction is impaired, constipation results. In certain states of the nervous system, the secretions of the mucous membrane and the contractile energy of the muscular fibres of the intestinal canal are both affected, with the result to render the intestines exceedingly torpid. Affections of the mucous membrane, disease of the liver and pancreas, the use of food leaving no residuum, over-action of the skin or kidneys, the state of anæmia or chlorosis, etc., lessen the proper action of the bowels by altering the character or reducing the amount of the proper secretions. The muscular contractility is impaired by a sedentary habit, by allowing the contents to accumulate, and disregarding the call to evacuate the canal, by the use of warm purgatives, and by a depressed state of the nervous system. As the condition of the trophic and vaso-motor systems must have a direct effect on secretion, it follows that constipation may be produced by all causes depressing these functions, as despondency, protracted mental activity, certain metallic poisons, notably lead, and diseases of the spinal cord.

Pathogeny and Symptoms.—The degree of fecal retention which constitutes constipation is not the same for all individuals. There are many—a majority, probably—who have a daily evacuation; others have two, and it is by no means uncommon for those in full health to have a movement on alternate days. After considerable observation, my conclusion is, that the tendency to constipation is less harmful than the opposite condition. There are instances in which a state of health has coexisted with a very considerable degree of constipation—with an occasional movement of the bowels—once a week, once in two or three weeks, or at longer intervals, indeed. Such instances are exceptional, but they are sufficiently numerous to justify the expression of the belief that the existence of constipation has not the deleterious effects often ascribed to it. Every individual must be regarded as a law unto himself, and his condition must be studied by the illumination afforded by his peculiarities in this respect. That which is a condition of constipation and of evil omen in one individual, may be a state of relaxation and of apprehension from this cause to another.

Constipation is accompanied by the symptomatic disturbances of the various maladies with which it is associated. We are here not concerned with these, but with the symptoms properly pertaining to the state of constipation. The tongue is usually coated, often heavily so, and the large papillæ at the base of the organ are swollen and prominent. The breath is heavy or fetid. The sense of taste is rather dulled, and the appetite is indifferent or voracious (bulimia). Very often the appearance and odor of cooked food excite a sense of disgust. When the stomach is empty, a feeling of depression is felt, and, after food, uneasiness, weight, and oppression. If any considerable fecal accumulation has occurred, the evidence may be afforded by the physical signs. Dullness of percussion along the course of the cæcum and colon, especially over the cæcum and above the sigmoid flexure (descending colon), will indicate the existence of fecal accumulation. Digital exploration of the rectum will disclose the amount of accumulation in that part of the canal. While, then, along the course of the colon a flat percussion note will be returned, around the umbilicus (over the small intestines) there may be a tympanitic sound. In cases of considerable accumulation, the whole abdomen may return, on percussion, a dull, even flat sound. The condition of other organs contributing to the constipation must be discussed elsewhere.

When the desire to have an evacuation is experienced, some pain is felt, and the movement is effected with straining and "bearing down." If the fæces have formed in a solid and dry mass, there may be, and often is, no little difficulty in effecting its expulsion. The *sphincter ani* is widely distended; the mucous membrane splits with the force of the expulsive effort acting on the solid fæces; more or less blood, mucus, and liquid fæces escape; and, finally, by a prodigious effort, only comparable to the pangs of labor, the hardened fæces are expelled in a large cylindrical mass. So severe the pain, so violent the expulsive effort, and so much injury done to the parts, that the system at large participates in the effects; the pulse becomes feeble, the face grows pale, and a clammy sweat bedews the surface. In some instances, especially in old persons having weak abdominal muscles, the contractile efforts of the muscles concerned in defecation are unequal to the task of expulsion, and hence the fæces, despite all their efforts, can not be propelled beyond the rectum, and then mechanical means become necessary to remove the accumulation.

The fact should not be overlooked that very considerable accumulations of solid fæces may take place, although apparently daily evacuation of rather liquid stools may occur. This paradoxical condition is due to the fact that the solid matters collect in the sacculi of the large intestine, leaving a central canal through which semi-solid and liquid materials may pass. It follows that with extensive fecal accumulation there may be daily, even more frequent, discharges. The author has encountered this state of things, more especially in old men, in whom a gradual and long-continued accumulation of the fæces has so lessened the perceptive and reflex activity of the mucous membrane, that hardly any extent of accumulation sufficed to excite the peristaltic action. Extensive accretion of fæces in the rectum has, in a few instances, been mistaken and treated for scirrhus. In women at all ages after adult life such fecal collections may form in the large intestine, especially in the cæcum and rectum. When, at last, the bowel contracts on its contents, very great pain is felt, the expulsive efforts are severe, and, finally, the accumulated mass is discharged.

More frequently, in cases of constipation, the retained fæces assume a globular shape (scybala), and are apt to be coated with mucus. When a movement of the bowels takes place, instead of a proper evacuation, there will be discharged a few of these round balls. The color of the fæces is, in simple cases of constipation, not altered; but when the torpor of the bowel is secondary to hepatic disease—to catarrhal jaundice, for example—the absence of bile-pigment makes a difference in the character of the stools.

In that form of constipation known as habitual, and in which the intestinal torpor is the substantive disorder, there are disturbances which properly belong to it. Fecal accumulation, by interfering with the return of blood from the inferior hæmorrhoidal veins, will cause them to swell, and, pushing before them the mucous membrane, will form hæmorrhoidal tumors or piles. By the same mechanism, congestion of the other pelvic viscera will be induced, and hence menorrhagia, catarrh of the uterus, dysmenorrhœa, etc., will result. In other instances fecal accumulations, by pressure on the nerves, may set up a degree of irritation sufficient to eventuate in a formidable sciatica, or, by pressure on the spermatic vein, to cause varicocele, or, on the ascending *vena cava*, to produce a varicose state of the veins in the inferior extremities.

The long-continued retention of hardened fæces has, finally, an effect on the mucous membrane; a catarrh is gradually set up, and, the irritation gathering strength with its persistence, a profuse diarrhœa ultimately results. It happens in this way that, in many cases of constipation of a persistent character, diarrhœa alternates with a suspension of action or constipation. For a time the bowels will be exceedingly torpid; then there will occur more or less pain, flatulence, tormina, etc., and a profuse diarrhœa; after which the ordinary torpor will come on, to be followed ultimately by an acute catarrh again. In some instances, but rather rarely, the irritation of hardened, retained fæces sets up a croupous inflammation, and thus to constipation will be added a membranous enteritis.

It is not always easy to separate the symptoms due to constipation from those of which constipation is a mere sign. Nevertheless, there are some clearly referable to this condition. When constipation has existed for some time, the nutrition is impaired. The intestinal digestion and absorption become languid, and hence the function of primary assimilation is inefficient. The result of this condition is shown in the thinness of the body and the diminished muscular activity. Under these circumstances, also, the products of intestinal digestion enter the blood in an imperfectly elaborated form. Various secondary disturbances occur. There are, for example, certain disorders of the skin produced in this way ; as, psoriasis, eczema, erythema, urticaria, lichen, etc. If the constipation and the causes producing it are removed, the cutaneous manifestations also disappear. Without the evidence afforded by a defined eruption, the functions of the skin are recognizably altered by the condition of constipation. The cutaneous secretions are deficient, the epidermis is dry and harsh, the circulation languid, so that the extremities are cold, and the hair and nails are brittle and wanting in a proper physiological activity.

There is an intimate association between the intestinal and cerebral functions. This fact was recognized by the ancients in the term *hypochondria*—under the ribs. They found that, with a certain depression of the feelings, with melancholy, there occurred disorders of intestinal digestion, flatulence, and constipation. They had also observed the good effects in certain mental disorders of the more active cathartics. As with the condition of constipation, and the consequently impaired primary assimilation and lessened excretion by the intestinal glands, various effete products accumulate in the blood, it follows that the cerebral organs must be affected by them. Hence follow headache, hebetude of mind, vertigo, etc. The eminent writer who prepared himself for his task by taking a brisk cathartic, practiced an eminently rational expedient. Stupor in some, an obstinate wakefulness in others, are also caused by constipation. As the general blood-pressure can be lowered by brisk purgation, it follows that the state of the intra-cranial circulation is affected by constipation.

Various circulatory disturbances are produced by constipation, and mention has been made of hæmorrhoids and hæmorrhages. When the general cavity of the abdomen has been filled out by retained fæces, the column of blood in the ascending vena cava is pressed upon, and hence the return of blood from the inferior extremities is impeded. It follows, from this mechanical obstruction, that œdema of the ankles takes place, and this result is the more apt to occur if the subject is obese, since the intra-abdominal pressure is the greater the more fat is contained in the omentum. Whenever the ankles swell, great anxiety is felt by the patient lest dropsy is about to develop.

Constipation also increases the secondary results of pulmonary obstruction. When, in consequence of mitral lesions or lung-diseases, the venous circulation labors, and the right cavities of the heart are overladen, fecal retention adds materially to the obstruction.

It follows, from the foregoing considerations, that constipation must be regarded from two points of view—as the sole cause of the morbid state, and as an important element in the pathological complexus.

Treatment.—Constipation is readily overcome for the time being by the administration of purgatives, but this practice can not be approved. The habitual use of purgatives—of purgative medicines and of purgative mineral waters—only increases the disability which they were intended to remove, for the bowels, becoming accustomed to the stimulation, will not act without it after a time. Before deciding on the treatment, a careful survey should be made in every case, and the causal condition should be ascertained, and efforts made to remove it. Is secretion insufficient? If this be causative, remedies to promote the secretions are necessary. If the motions indicate the absence of bile, phosphate and sulphate of soda and sulphate of manganese will be found effective; the last, especially, if there be a gouty habit; and the first two combined or not with arseniate of soda, if there be a tendency to contraction of the liver (cirrhosis). If the state of constipation be habitual, associated with paresis of the muscular layer of the bowel, and also deficient secretion, the best results may be expected from physostigma (tincture), nux vomica (tincture), and belladonna (tincture), in combination, and the action of these remedies may be promoted by combination with aloes (tincture) when the lower bowel is torpid. To these remedies must be added galvanic and faradic stimulation, or the combined currents, which seem to be more efficient.

If the subject is plethoric, the secretions deficient, the muscular layer wanting in contractile energy, a combination of magnesia sulphate (Epsom salts) with diluted sulphuric acid and sulphate of strychnine (one sixtieth of a grain), in solution well diluted, may prove highly effective. The addition of sulphate of iron renders the just-mentioned formula very useful in anæmic subjects, suffering under the same condition of the digestive tube, or in those of relaxed habit.

When we have to deal merely with paresis of the muscular layer of the bowels, the remedies must consist of those having the power to increase the contractile energy of this part. Nux vomica, belladonna, and the warm purgatives, have this effect; and hence a combination of these may be very useful. To these must be added electricity. One electrode is placed in the rectum—an insulated electrode, with a metal button or bulb uncovered—and the other, in the form of a moistened sponge, passed over the abdomen, especially along the course of the large intestine. Daily applications of a faradic current, or of a slowly-interrupted galvanic current, made as just described, will often effect permanent relief. Other forms of stimulation of the bowel may be good in these cases of torpor dependent on paresis of the muscular layer. A nightly dose of podophyllin resin, with the extracts of belladonna, nux vomica, and ergot, may establish a regular habit; but the concurrent action of certain hygienic measures may be necessary. The author has known of excellent results from the regular use of confection of senna every night on retiring, the quantity being very slowly reduced as the habit of a daily movement is effected. This preparation may be increased in activity by the addition of a minute quantity of podophyllin resin, or croton oil. Other substitutes are cascara sagrada taken on going to bed, minute doses of podophyllin resin at the same time and regularly, and both of these have strong reports in their favor.

Hygienic means are not less important than the medicinal in the treatment of constipation. Regularity of habit should be inculcated. The patient should be instructed to repair to the closet at a fixed hour, selected with reference to convenience and leisure especially. An effort should be made, whether the desire be present or not. At the same time the abdomen should be thoroughly kneaded and rubbed. A large draught of cold water should be swallowed before breakfast, unless some contraindication exist. This may be sufficient, if a little salt—a teaspoonful—be added to the water. In the torpor of the bowels belonging to old people, a morning-draught of an alkaline mineral water—Saratoga, for example—has an excellent effect. In the case of the plethoric suffering from constipation, the purgative waters—Saratoga, Pullna, Hunyadi, and others—taken in the early morning, have an unquestionable good effect. Above all other means for removing constipation are those hygienic appliances derived from the natural stimulus of the intestinal movements—food. If there be no contraindication, those foods which leave a considerable residuum—as Graham flour, rye and corn bread, oatmeal, cracked wheat, etc.—can be used with distinct advantage; fresh vegetables of the succulent class—as lettuce, spinach, celery, onions, etc.—and fruits—as apples, dried peaches, figs, dates, tamarinds, etc.

As a sedentary life induces constipation, it follows that exercise must be enjoined in such cases. Mere enforced exercise is not so beneficial as that kind of active movement necessitated by travel. The author has seen some cases of constipation cured by a trip to Europe. Horseback exercise is an excellent mode of progression when practicable; but walking may always be carried out efficiently, and must, therefore, be chiefly depended on. The best results are obtained by a proper combination of the various curative agencies.

OBSTRUCTION OF THE INTESTINES.

Definition.—By obstruction or occlusion of the intestines is meant an arrest of the passage of their contents, by obstacles within the bowel, or in its walls, or in the cavity of the peritoneum. When the obstruction occurs in the intestine after it has passed out of the cavity—as strangulated hernia, for example—it becomes a surgical malady. A great many names have been applied to this state: ileus, iliac passion, volvulus, miserere, etc.

Causes.—Obstruction or occlusion of the intestines may be produced by causes that are intrinsic, or extrinsic, but they are best considered in three great divisions: 1. Extrinsic, or entirely outside of the bowel; 2. Conditions affecting the walls of the intestines; 3. Disorders within the canal.

1. The extrinsic causes are tumors without, compressing the intestine; certain orifices in the peritoneum, as the foramen of Winslow; bands of connective tissue, remains of former inflammation; twisting, or torsion, of the bowel.

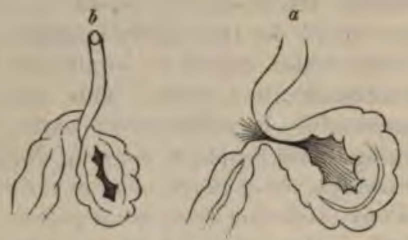

Fig. 3.—Mode in which twisting of the bowel occurs. *a*, The first derangement. *b*, The twist.

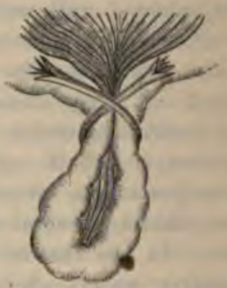

Fig. 4.—Constriction by a band of lymph.

The tumors coming into relation with the intestine, and obstructing by pressure, are of various kinds: floating kidney, displaced spleen, cysts of the peritoneum, tumors of the mesentery, of the ovary, etc., and cancer in various situations. As regards the entanglement of the bowel by passing into certain orifices, especially the foramen of Winslow, the accident is rare (three cases recorded), but a number of examples have now been noted of retro-peritoneal hernia, first accurately described by Treitz.* The duodeno-jejunal flexure is embraced in a fossa formed by a fold of peritoneum, "continuous on its inner side with the peritoneum covering the transverse duodenum, and forming the inferior layer of the transverse mesocolon." Diaphragmatic hernia is relatively more common; Leichtenstern collected two hundred and fifty-two cases. There are certain weak points in the diaphragm—at the œsophageal foramen, just behind the sternum, the space between the lumbar and costal parts of the muscle of the diaphragm—through which parts of the bowel and omentum have passed.

Constriction by old bands of adhesion, the result of former inflammations, is much more common than the herniary protrusions. The adhesion of the appendix vermiformis to the abdominal wall, or to neighboring parts of the intestine, forms a transverse band in which a knuckle of intestine may become engaged. Similar bands, or bridges, form between the organs in the pelvic cavity, and between the mesentery and intestine. Some of these bands, owing to changes made by the movements of organs, often quite considerable, attain to great lengths and form constricting loops of various kinds. Slits are found in the mesentery, especially in the mesentery of the ileum, and low down, into which a fold of the intestine may drop and become incarcerated. The extremity of diverticula becoming attached by bands of lymph, also form openings into which the intestine may pass. There is, indeed, almost no limit to the forms and varieties of constricting bands for the occlusion of some part of the intestine.

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* Dr. P. H. Pye-Smith, "Guy's Hospital Reports," third series, vol. xvi, p. 131, "On Retro-peritoneal Hernia."

Also, occlusion may be brought about by twisting (torsion) of the bowels. The sigmoid flexure is especially liable to this accident, owing to its shape and to congenital defects, and next the cæcum; rarely does this accident happen to any other part of the canal. I owe to Dr. Starck, of this city, the details of a case of this kind, in which two inches of the upper part of the ascending colon slipped over the part below. In the preliminary changes which occur in the sigmoid flexure preparatory to torsion, the mesenterial root shrinks and the two ends of the fold approximate, so that twisting can easily occur if the peripheral part of the fold is full of fæces and therefore heavy. The length and weight of the fold prevent untwisting, while rapid swelling and distention by gas, occurring in that part of the bowel above, keep the fold in position.*

While twisting of the sigmoid flexure is apt to take place in early life, torsion, or twisting, of the cæcum is a malady of advanced life rather—in more than half of the cases occurring from forty-five to sixty years. Owing to the changes produced by old hernias, to the absorption of fat in the mesentery, and to paresis of the muscular layer with resulting accumulation of fæces, a loop of the cæcum and ascending colon forms—with a contracted mesentery—the axis of the loop; the two ends of the loop approximate, and a twist may be readily induced by various forces, as sudden movements of the body, an abnormally long and full ileum, etc.

2. Changes occurring within the intestinal tunics, such as tumors, polypi, hydatid cysts, carcinoma, etc., cause occlusion by a gradual obliteration of the canal. More frequently is the obstruction due to cicatrices, formed by the closure of ulcers, notably those of dysentery, of typhoid fever, of syphilis, etc. The most important of this group of causes is intussusception. By this term is meant the slipping of one part of the intestine into the adjacent part, so that the peritoneal and mucous surfaces are opposed to each other. This accident always occurs from above downward. Frequently, after death, there are found invaginations, which formed during the last moments of life, but they have no importance. Often a number of them exist at various points.

As the part first invaginated remains at the point where it entered, it is obvious that the increase of the intussusception is by a continued slipping-up of the part below. The accident of invagination may take place at any point of the intestines, but the most common is that of the ileum into the cæcum, and this attains the greatest dimensions. In children the ileum may pass into the whole length of the colon, and be felt in the rectum and even pass through the anus. Other forms are of the ileum entirely, of the jejunum into the ileum, of the duodenum into the jejunum, of the colon, etc. Of all the forms of obstruction in the intestinal canal occurring in early life, that of invagination is most usual. Including all ages, half of the cases of intussusception occur before ten. As regards sex, males are more subject to the accident than females. There are two important elements in the mechanism—paresis, or distention, of a part of the intestine below; spasm, or contraction, of the part above. When the bowel is undergoing irritation and is distended with gas, if, in consequence of the same irritation, violent reflex contraction of the circular fibers is induced, it is not difficult to conceive of the suddenly narrowed portion dropping into the distended. Especially can we conceive this accident happening if the muscular layer of the enlarged portion of the bowel is in a paretic state, and the muscular layer in the narrowed part is in a tetanic or spasmodic state. A different explanation of the mechanism is made by others, especially by Leichtenstern, who affirms that there are two factors involved—a paretic condition of a part of the bowel; violent peristaltic action. He supposes that the invagination occurs entirely by an inversion of the paretic part of the bowel, and that this inversion is initiated by the excited peristaltic action. The differences of opinion are not very wide, after all, and are rather in the interpretation of terms than of the pathological factors. When intussusception occurs at the cæcum, doubtless the same causes are at work as those which induce protrusion of the bowel in dysentery—a violent tenesmus with paresis of the muscular layer—a condition of things which may readily arise in the ileum and the cæcum. When invagination has occurred, the mesentery being drawn in with the bowel and more or less stretched, the circulation is greatly impeded, especially the return of venous blood. Swelling ensues; the tunics of the invaginated portion of the bowel are infiltrated with bloody serum; an active catarrh of the mucous membrane is established; the peritoneum becomes intensely hyperæmic, and an abundant exudation is poured out, gluing together the contiguous portions of mucous membrane. In these cases there is not, necessarily, a complete occlusion—there may be still space for the passage of liquid fæces. The compression of the mesenteric vessels induces necrosis of the invaginated portion, which may slough off, and thus restore continuity.* It is necessary to this result that the invagination be equal on all sides, that union take place in a uniform manner around the bowel. If the invagination is unequal and the line of union irregular after the slough separates, in the course of contraction of the cicatrix which subsequently takes place, there may be produced very considerable deformity of the intestine, and its lumen seriously encroached upon. Again, when the slough separates, the adhesion may be insufficient, thus opening into the general cavity of the peritoneum.

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* Dr. Küttner, in St. Petersburg. Virchow's "Archiv," vol. xliii, p. 478, "Ueber innere Incarcerationen." A full account of the subject, with admirable plates showing the mechanism of twisting. Ibid., Band liv, S. 34. Also in the same, "A Case of Internal Strangulation," by Jacob Heiberg, with two illustrative diagrams.

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* Trousseau, "Clinique Médicale," tome iii, p. 196. He has had two cases of this kind.

3. Causes of obstruction within the canal of the intestines (intrinsic) are quite frequent—relatively more so than the extrinsic causes. First in importance is fecal accumulation, forming most frequently in the cæcum and ascending colon, and in the descending colon just above the sigmoid flexure. Not unfrequently such fecal accumulation has for a nucleus an intestinal or biliary calculus. The intestinal calculi are composed of ammoniaco-magnesian phosphate, and the carbonate and phosphate of lime, with more or less inspissated mucus (enteroliths). Other foreign bodies accidentally present in the canal may form a nucleus about which the salts above named crystallize or adhere. They are usually oval in shape, but may have a great variety of forms, and they differ in size, the average being about the size of a chestnut. Large concretions of chalk and magnesia have formed when these substances had been taken medicinally for some time. Stones of such great size have formed, that they are alone sufficient to cause obstruction. The usual results of their presence, if they occasion symptoms, are attacks of intestinal indigestion, colic, typhlitis, ulceration, and perforation of the cæcum and appendix. Biliary calculi much more frequently occasion obstruction ; although of considerable size, they have been passed without any trouble. Sometimes, the symptoms of acute intestinal catarrh, pain, flatulence, nausea, diarrhœa, etc., are caused by them ; again, the bowels are obstructed more or less completely by one, or a succession of attacks of impaction, relief from one attack being followed in a few weeks by another attack of the same character, have been produced by a gall-stone, lodging successively in different parts of the ilium. Now and then complete obstruction has been caused by a gall-stone. They occasionally set up an ulcerative process in the cæcum and appendix. An important factor in causing obstruction of the bowel is habitual constipation—that form, especially, which consists in a paretic condition of the muscular layer, and a state of diminished sensibility of the mucous membrane. Abnormal flexures of the colon often play an important part in causing an obstinate constipation. Accumulations occur to a very great extent behind the natural and factitious flexures, and in the cæcum in old subjects especially, in women leading very sedentary lives, and very careless. Large accumulations are not incompatible with daily, even more frequent evacuations. The central canal may still continue open and yet enormous

masses remain in the sacculi. Finally, some large fecal masses drop into the canal, and symptoms of occlusion at once appear.

Symptoms.—The cause and the seat of the occlusion affect somewhat the character and development of the symptoms, but there are certain symptoms common to all forms: these are pain, arrest of the intestinal movements, gaseous distention of the bowels, and vomiting. The pain is not acute and lancinating, but is severe, colic-like, with a feeling of soreness, and is aggravated by pressure. In the beginning the pain is felt about the umbilicus, in the iliac regions, and radiates thence over the abdomen. When tenderness to pressure exists at the outset, it is indicative of the seat of the lesion, but the tenderness is rather a feeling of soreness, and has not the painful character of the tenderness which is developed later on when peritonitis appears. It is important to note that the tenderness and pain cease when collapse comes on—for the author has known this to be mistaken for improvement. At first, and usually after the administration of an enema, there may be an evacuation from the lower bowel, and this is often a source of misapprehension, for it is assumed that the canal is not obstructed. It may be regarded as an evidence that the obstruction is above the sigmoid flexure, but it has no higher significance than this. At the beginning of symptoms—of intussusception, for example—some liquid fæces may escape, but presently the obstacle to the passage of fecal matters and of gas is complete. Even when those exceptional discharges, just referred to, escape, there is no improvement in the feelings or condition of the patient; they do not diminish the fullness and tension of the abdomen. When complete obstruction has existed twenty-four to forty-eight hours, the abdomen is no longer soft and flexible, but the muscles have become rather rigid, and the whole abdomen is swollen and hard, returning on percussion a note of tympanitic quality, except where an accumulation of fæces gives a different tone. In the further progress of the case, more and more gas distending the intestines, they can be distinguished as inflated, sinuous cylinders: the small intestines filling the umbilical space, the large intestine, the flanks, and the lower epigastric region. Not unfrequently the abdomen is uniformly distended, the highest point in the centre and falling off in all directions, and the walls drawn as tense as the tightened drum-head. Besides the immediate and local distress thus occasioned, the functions of the thoracic organs are interfered with by the upward pressure. The respiration is thoracic, oppressed, and hurried, a distressing hiccough supervenes, and the action of the heart is troubled. Vomiting is a most characteristic symptom under certain circumstances. It sometimes begins early, immediately after the obstruction, and consists at first of aliment, then of mucus, mucus and gastric juice, mucus and bile from the gall-bladder forced up by the straining. On the other hand, vomiting may be postponed until the signs of obstruction are well advanced. If vomiting persists, presently the matters returned consist not only of greenish sero-mucus, but of the contents of the lower ilium, and having a fecal odor. Indeed, distinctly formed but not molded fæces have been returned by vomiting, but usually it is a yellowish fluid, having the consistence of soup, and an odor and taste sufficiently definite. The fecal vomiting recurs from time to time, and, if it well empties the intestines of their contents, the abdominal symptoms are improved; there is much less distress, and the distention is diminished, so that the thoracic organs are not so embarrassed, but this merely local improvement does not help the case otherwise. The gravity of the case is illustrated in the systemic condition, which becomes rapidly bad. There is no fever, but a temperature below rather than above the normal. The countenance at first expresses great anxiety, then becomes contracted and drawn, the eyes deeply sunken and surrounded with a livid circle, the nose pinched and blue, the lips blue, the tongue dry, the voice husky and sepulchral, the surface of the body generally cold and covered with a cold sweat, the skin livid and wrinkled, hiccough persisting and more and more harassing, the breathing more shallow and rapid, the temperature declining a degree or two Fahr.—such is the complexus of symptoms in the approaching collapse. Usually the mind is clear and the anxiety great, but there may be an inexplicable apathy, and in rare cases acute delirium. Toward the close, the increasing difficulty in hæmatosis develops carbonic-acid poisoning, and then stupor ensues. The symptoms of occlusion, due to invagination, differ somewhat from the other forms of obstruction, and must therefore receive attention. The attack usually sets in suddenly as the intussusception occurs quickly, and the first symptom is violent, colic-like pain, which is followed by vomiting, the more prompt and certain the nearer the trouble is to the stomach. In children the first colic-attack is followed after a few hours by relief, which continues for several hours until a new seizure; but in the case of adults the pain which marks the occurrence of the intussusception continues for several days, after which it is paroxysmal, there being intervals of exemption from suffering. A very troublesome diarrhœa is coincident with the invagination, from ten to twenty, or even thirty discharges occurring daily, and these soon assume a dysenteric character, owing to the intense congestion of the intestine at the point of invagination. This symptom has greater significance, because no other form of occlusion of the bowel presents it. The tenesmus is all the more severe when the bowel descends into the rectum, as it sometimes does in children, and with this condition may be associated involuntary discharges of mucus and blood, because of paresis of the sphincter ani. There may be considerable variation in the meteorism in invagination—great distention occurring immediately after the accident has occurred, then subsiding as the diarrhœa goes on. A cylindrical, soft, yet somewhat resisting tumor can often be detected on palpation, when the invaginations are in certain places: in the cæcum, transverse and descending colon, and at the sigmoid flexure. It is especially in children and in the chronic cases that these invagination tumors can be detected. There are peculiarities about these tumors which should be noted: they change in position somewhat, and in form, under the influence of peristaltic movements excited by the necessary palpation, or occurring spontaneously. In children the descent of the ilium is so very rapid that the rectum may be reached on the second day. An intussusception may induce obstruction at once, and death occurs in from three to six days, partly by exhaustion, partly by the local inflammation. In other cases, after the immediate closure of the bowel, the canal is partly restored by a subsidence of the local congestion, or the obstruction has at no time been complete: diarrhœa of an exhausting kind comes on; gangrene of the invaginated portion takes place; and in children death ensues from the fourth to the seventh day, but in adults the fatal result is postponed to the second, third, and fourth week, according to the acuteness of the symptoms. When, in the process of separation of the invaginated portion of the bowel already described, the discharge of the gangrenous parts takes place, it does not always occur in its entirety, but shreds and masses of various sizes are cast off, so that, indeed, the fact of such sloughs being present in the evacuations may escape detection. In the only case of invagination in which the bowel itself sloughed off in its entirety, in the practice of the author, the lost piece, a part of the ilium, was eight inches in length, entire as respects the presence of all the layers of the bowel, and showing the evidences of gangrene only at the line of separation. This occurred on the eighteenth day of the disease, the patient recovering. Again, cases of intussusception become chronic, last for months, even for a year or two, and then recovery ensues, or death takes place by gangrene, by perforation, by peritonitis, or by all of these accidents combined.

Diagnosis.—The diagnosis involves the two questions—1. Of the form of disease causing obstruction; 2. Of the seat of the obstruction.

1. *Form of Obstruction.*—This is usually a matter of inference; nevertheless, there are considerations which may conduct the observer to right conclusions. Palpation and inspection of the rectum may determine the existence of a tumor, an enterolith, or fecal accumulation. Fecal accumulations may also be distinguished by palpation at the sigmoid flexure and at the cæcum, and the diagnosis may be aided by the history of constipation. The occurrence of previous attacks of hepatic colic, if within a reasonable period, would be a presumption in favor of obstruction caused directly by a biliary calculus, or of impaction, the calculus serving as a nucleus for the formation of fecal masses. If attacks of typhlitis, of pelvic peritonitis, or of peritonitis

in other situations have occurred before, it may be that a knuckle of intestine has been fastened by such a band. If a floating kidney or other tumor has been known to exist in a situation to compress the bowel, when sudden occlusion occurs, the cause will be at once suspected.

2. *Seat of Obstruction.*—The diagnosis of the position at which obstruction has occurred is a little less uncertain than the determination of the form of disease.

The distention of the abdomen—the meteorism—may furnish valuable diagnostic indication. When the colon at its lower part is obstructed the rectum will be empty, but the transverse and ascending colon will form a prominent roll, the rest of the abdomen being relatively sunken. Ultimately the stretching of the large bowel will render the ileo-cæcal orifice incompetent, and then the small intestines will be inflated and the whole abdomen swollen. When, as is so frequently the case, the obstruction is at the ileo-cæcal valve, the whole of the large intestine will be empty, and then the flanks, and the epigastrium will be relatively flat and sunken, while the center of the abdomen, all around the umbilicus, will be prominent and distended. By palpation and percussion the situation of a tumor, or of a fecal accumulation, can be made out.

When obstruction occurs in the jejunum or duodenum, the course downward into collapse is more rapid, the vomiting and hiccough more persistent and exhausting than when the same obstruction exists at other points. Furthermore, the abdomen is not distended, may be retracted even, and the vomited matters contain no fæces. The urine is scanty in obstructions high up, and plentiful when the obstacle is low down in the colon.

If the symptoms have occurred suddenly, and are very acute, especially if peritonitis is present, a tight strangulation is probable—behind a band, in a slit in the omentum, or beneath the attached appendix.* If acute symptoms of obstruction have set in after some violent muscular efforts—as jumping—the patient previously free from disease, a twist in a loop of intestine has probably taken place. Has blood passed by stool in a child who has suffered from diarrhœa, and the symptoms of occlusion have come on suddenly, intussusception is the most probable nature of the accident. Whenever symptoms of obstruction occur in a woman who has borne many children, or is the subject of external hernia, or in one who has had attacks of peritonitis, the existence of strangulation by bands of adhesion is very probable.†

Course, Duration, and Termination.—All of these points have been more or less discussed, but some additional observations may be neces-

* Bryant, "The Medical Times and Gazette," vol. i, 1872, p. 363.

† J. Hutchinson, ibid., vol. i, 1858, p. 34.

sary. The various occlusions, even when they have existed to a partial extent for a long time, begin suddenly and with violent symptoms; their course is rapid, and they terminate in recovery, in partial recovery, in peritonitis, with or without perforation or gangrene. Peritonitis is a common result. It is announced by greater fullness of the abdomen, increased embarrassment of breathing, more frequent vomiting and hiccough, rise of temperature, and deepening of the collapse. The duration in the average is, according to Leichtenstern, six days; but a child may be killed by the shock of an intussusception in a few hours. They may last two or three weeks.

Prognosis.—In every case of occlusion the prognosis is grave; for, although even very unpromising cases may yield to treatment, the result is so usually fatal that the most guarded opinions only should be given. The prognosis is more favorable in cases of impaction by fæces than in any other form of obstruction.

Treatment.—Until the character of the obstruction is ascertained, no attempt should be made to procure a movement of the bowels by active purgatives or by enemata. If impaction be discovered, the treatment already described should be put in force. If intussusception be the cause of obstruction, then certain kinds of enemata are used. Nevertheless, the rule holds good that in obstruction all violent and perturbing measures are improper. On the other hand, the utmost quietude is necessary, in respect to the movements of the patient as well as to the use of remedies. Foremost, and above all measures, stands opium, administered with the view to maintain a quiescent state of the intestinal canal, and not less for its influence over the inflammation and spasm which arise in the course of the various obstructions. The most effective mode of administration is by the hypodermatic injection of morphine. The quantity is measured solely by the effect produced. There should be sufficient morphine administered to quiet the pain, to lower the pulse, and to maintain a state of somnolence from which the patient may be easily aroused. This is accomplished in adults by one fourth of a grain of morphine and $\frac{1}{120}$ grain of atropine for the first injection, and by one eighth of a grain subsequently, and every four to six hours, according to the degree of effect. With each subsequent dose from the first, the quantity of atropine should not be greater than $\frac{1}{200}$ of a grain, for the effect is much longer maintained than is the case with morphine. When impaction exists, the use of the opium would seem not to be indicated, since constipation is a leading factor, but even in these cases the result of its administration is much more favorable than the treatment by purgatives, which in vain are used to overcome the obstacle; while, if the opium be persisted in, the bowels move spontaneously. Purgatives failing to remove a fecal accumulation, an invagination, or internal strangulation, increase all the dangers—of gangrene, of perforation, and of peritonitis. Although this agent is more effective when used in the form of morphine subcutaneously, various preparations of the crude drug may be administered by the stomach or by the rectum, the object in view being the same. Next to the subcutaneous method, probably the most satisfactory mode of administration is by the rectum. For stomachal use the best preparation is the official deodorized tincture.

If the meteorism be very pronounced, this increases the difficulty of relieving the invagination or the internal strangulation by maintaining an over-distention of the intestine above the point obstructed. The gas may be safely removed by puncture with a fine, long needle of the aspirator. This little operation, by removing an accumulation of gas, has permitted the reduction of strangulated hernia, which had previously resisted the most skillful taxis. Experience has abundantly shown that the distended intestines may be punctured at various points without any ill result, immediate or remote.* An intussusception through the ileo-cæcal valve or an impaction of the cæcum and ascending colon may now and then be overcome by hydrostatic pressure—by filling the intestine gradually with water at 95° from a reservoir placed at a sufficient elevation. Air or gas may be used for the same purpose. A neat way to effect it is to disengage carbonic-acid gas in the rectum by injecting first a solution of sodium bicarbonate, and following this with a solution of tartaric acid. About a drachm of each will be required. A firm compress must be held against the anus with sufficient strength to prevent the escape of the gas. Such is the elastic force of the gas that the intestine is distended, the ileo-cæcal orifice expanded, and the intruded bowel forced back. For the success and safety of this expedient it is essential that it be used before peritoneal exudation and adhesions have formed—before, indeed, the intruded bowel is much swollen. If put off too long, adhesions, to prevent rupture into the peritoneal cavity, may be destroyed, or a softened condition of the bowel will yield before the pressure of the gas and a rent occur. For these and other reasons an experiment of this kind should be undertaken early. The distention of the bowel by air forced in by an ordinary pump may be used instead of gas, or tobacco-smoke may be injected, partly to act mechanically, partly as a relaxing agent. The infusion of tobacco was formerly much employed, but rarely now, as an enema to relax the muscular fiber of the intestine. It is a very dangerous application, and is not as effective as other means now used. Recently irrigation of the stomach has been warmly advocated. It is alleged that, by thoroughly emptying the bowel of gas and fæces through the stomach-tube, the distresses of the patient are much mitigated, and the obstruction, if due to impaction, invagination, or twisting, is overcome.

Warm applications to the abdomen afford comfort, if they do not affect the course of the disease. If there be local tenderness—in the right iliac fossa, for example—an ice-bag may be placed over the painful spot, and, if the temperature is elevated, leeches may be used cautiously. Whenever, in intestinal maladies, leeches are to be applied, the anal region should be selected. As the strength of the patient is rapidly reduced, much attention should be paid to alimentation. Solid food should not be given. Milk, eggs, and meat-juice are proper. If vomiting persists, lime-water should be added to the milk. Champagne and cracked ice are highly grateful to the patient, and allay vomiting. Stimulants are required as the symptoms of collapse appear. Carbolic acid in mint and cherry-laurel waters is useful to allay nausea and to remove the fetor of stercoraceous vomiting. The author is aware that many practitioners administer various agents in combination with opium, partly to increase its efficacy, it is supposed, and partly on account of some virtue in the remedy. Calomel is most frequently so employed, and, as the author believes, to the injury of the patient, except when given in very minute doses to allay irritability of the stomach. The relief of internal strangulation, by surgical methods, does not come within the scope of a strictly medical treatise. The reader is referred to papers by Mason and Ashhurst.*

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* Trousseau, "Clinique Médicale," *op. cit.*

INTESTINAL PARASITES.

Forms.—Only those parasites having their *habitat* in the intestinal canal will be considered. Trichinosis, the most important subject in helminthiasis, pertains to the class of general diseases, and will therefore be treated of in that connection.

But twenty-one of the large number of parasites infesting the human body are found in the intestinal canal, and of these only eight are peculiar to man. They are as follows:

Cestoda (Tape-worms):	Tænia solium, Tænia saginata, Bothriocephalus latus.
Nematoda (Round Worms):	Ascaris lumbricoides, Oxyuris vermicularis, Trichocephalus dispar, Trichina spiralis, Anchylostomum duodenale.

One parasite at a time is the rule—two is not an uncommon number; but Rosen † reports the case of a child four years of age in whose intestines there were ten lumbricoid worms, an innumerable quantity of oxyures, and four tæniæ. According to Davaine, ‡ children are more affected by nematoda (round worms), and adults by cestoda (tape-worms), but Heller[*] maintains that adults are more affected by both classes of parasites.

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* "The American Journal of Medical Sciences," 1873 and 1874, vols. lxvi and lxviii.

† "Traité des Entozoaires et des mal. Verm.," par C. Davaine. Paris, 1879.

‡ Ibid.

Origin.—The doctrine of spontaneous generation having received its fatal blow, it is unnecessary to discuss this theory as applied to intestinal worms. It may be regarded as settled that the ova or embryos are admitted from without and conveyed into the intestinal canal by articles of food and drink. Hence, those who handle fresh meats or eat uncooked animal food are specially liable to become hosts of parasites.[†] Uncleanliness is also an influential factor, and for obvious reasons.

General Results of the Presence of Parasites in the Intestinal Canal.—There is scarcely a symptom which has not been referred to worms. Formerly, as an etiological factor, worms had a high degree of importance; but their influence has been less and less regarded, so that now they are almost wholly overlooked. As is usual, doubtless, the truth lies between these extremes. The presence of parasites in the intestinal canal is not incompatible with perfect health and the entire absence of symptoms. The effects produced are local and systemic. The local symptoms are, disorders of digestion, abdominal pains, especially around the umbilicus, and an irritation, usually an itching, around the anus; but the chief symptom is the appearance of the worm or worms. The remote or systemic signs are very numerous: thirst; salivation; a capricious, absent, or exaggerated appetite; emaciation; irregular action of the heart, palpitations, or intermittence of the pulse; cough, dyspnœa, laryngismus stridulus; disorders of taste, hearing, smell, vision; convulsions—such are the varied reflex disturbances produced by parasites in the intestinal canal. They are, however, far from usual; indeed, they are exceptional, and not determined by the size, number, character, or position of the worms, but on some special susceptibility of the affected person.

CESTODA—TÆNIA—TAPE-WORMS.

Varieties.—Tænia saginata—beef tape-worm—is the form most common in this country; tænia solium is occasionally encountered, while the bothriocephalus latus is rare.

Causes.—The development of tænia in its different phases has now been thoroughly demonstrated. Bothriocephalus latus has, however, thus far eluded research. A tape-worm reaches its final growth in the intestinal canal from an embryo—an intermediate stage in its course of development—admitted into the canal by means of infested meat. Since the introduction of the Russian method of curing diarrhœa by the use of finely-scraped raw meat, and the modern taste of eating

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[*] "Intestinal Parasites," *loc. cit.*

[†] Cobbold, "Entozoa." London, 1864, p. 232.

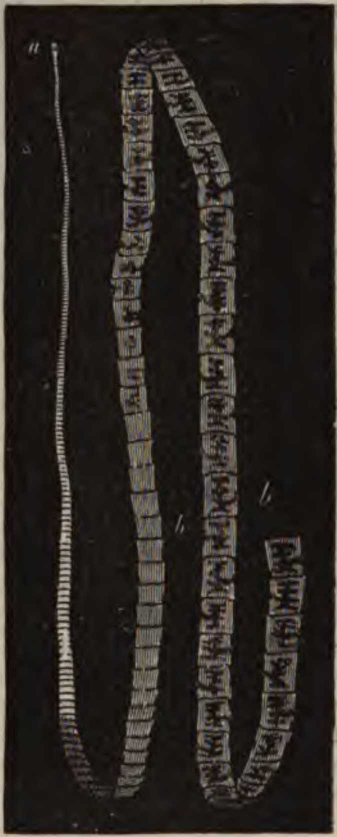

Fig. 5.—*Tænia solium*, or solitary worm. *a*, head, or scolex; *b*, tape formed of many individuals, the last of which, completely sexual, separate under the name of ***proglottides***, and represent the adult and complete animal. Each solitary worm is a colony.—*Van Beneden.*

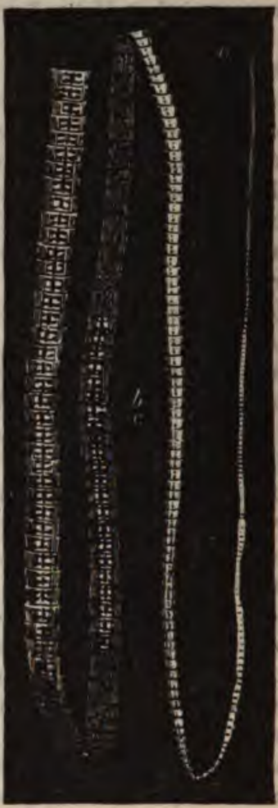

Fig. 7.—Bothriocephalus latus. *a*, scolex; *b*, the proglottides; *c*, the sexual organs.—*Van Beneden.*

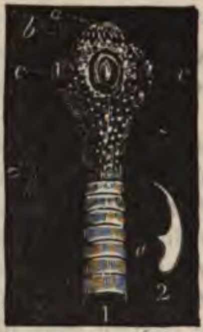

Fig. 6.—*a*, Rostellum; *b*, crown of hooks; *c. c*, suckers; 1, scolex of the tænia solium; 2, hooks expanded; *a*, heel of the hook.—*Van Beneden.*

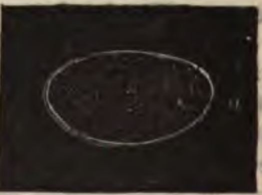

Fig. 8.—Bothriocephalus latus, egg.—*Van Beneden.*

rare steaks, etc., tape-worm has become more common. Tænia solium is derived from the embryos contained in pork, known as *cysticercus cellulosus*, and T. saginata, from embryos found in beef. The bothriocephalus is supposed to be derived from an embryo found in fish, but not correctly so, as it occurs among peoples living on the seashore and at the borders of lakes, and in the interior of continents as well.

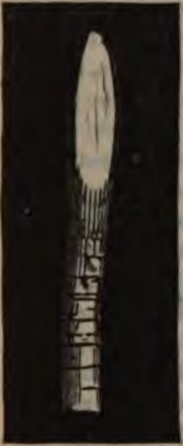

FIG. 9.—Bothriocephalus latus, scolex.

Symptoms and Results.—The small intestine is the abode of tænia, but when very long it may reach into the large intestine. The head is fixed against the mucous membrane just below the pylorus. The T. solium is usually solitary, but not always, and a number of them may be found in one host. The immense length of the segments discharged often gives rise to the impression that there must be several of them to produce such a quantity. Although more frequent in adults, no age is exempt, and infants at the breast have been infested after feeding on raw beef-pulp. Dr. Armor* reports a case of tænia in an infant five days old. Women are more subject than men to tænia: in one hundred and sixty-four cases, ninety belonged to women and seventy-four to males. Segments or strobila of the tape-worm colony pass in numbers spontaneously, and after the action of medicines; and now and then the living proglottides migrate, crawl out of the anus, and are felt, cool and moist, wriggling about the hips, thighs, and genitals. Very rarely, portions of a tape-worm are thrown up by vomiting. The length of time they remain in the intestine is by no means a fixed period; they have been known to exist there ten to twelve years, and even longer; but there are very obvious difficulties in the way of accurate determination of this point.

The presence of a tape-worm when recognized by the patient induces serious inquietude of mind, but not necessarily any disturbance of the bodily functions. Not unfrequently, a tape-worm produces, absolutely, no symptoms. The degree of disturbance caused is determined by the characteristics of the affected person—they who suffer much are nervous and easily susceptible to impressions of all kinds. In a large proportion of cases, the presence of the proglottides in the evacuations is the first intimation of the presence of the worm in the intestinal canal. The principal symptoms are: emaciation, notwithstanding an inordinate appetite; a feeling of lassitude; colicky pains felt through the abdomen; palpitation of the heart, faintness; salivation; disordered digestion; pruritus of the anus and nose; disorders of the special senses, notably feebleness, etc. Sometimes the disagreeable feelings in

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* "New York Medical Journal," December, 1871.

the abdomen are removed by taking food. Probably the most constant symptom is the colicky pains felt in different parts of the abdomen; but they are not always present, are intermittent, and vary as much in severity as in situation. Constipation is more usual than diarrhœa, and they may alternate. Itching about the anus and nose is a common symptom, and is rarely absent from one or the other situation, but itching of the anus is more frequent. The nervous phenomena, strictly speaking, are very pronounced, consisting of affections of the special senses, pains and cramps in the extremities, choreic seizures, epileptiform attacks, hysteria, etc. In a few cases the patients experienced a horrible odor, purely subjective; others have disagreeable sensations excited by music; others have impaired vision, sometimes complete amaurosis, now affecting one eye, now the other; again, there are those who have, instead of itching, a sensation of hyperæsthesia or anæsthesia in certain parts of the body, a momentary loss of voice or of memory, persistent wakefulness, epistaxis, etc. The most important symptom is the passage of strobila, or, more frequently, proglottides. Each proglottis contains the sexual apparatus complete and a multitude of embryos, and has a power of motion when first detached from the strobila or tape-worm colony. It is then a segment—a moist, whitish, cool, quadrangular body, like a bit of stout white tape, but changing its shape constantly so long as the power of motion lasts. Inspected with an ordinary pocket lens, the uterus and ovisacs, with their lateral branches on one side, and the testicular bodies on the other side, can readily be seen. It is quite possible to differentiate between the T. solium and T. saginata by an inspection of the proglottides—the former being thinner, softer, and more transparent. The lateral branches of the uterus of the T. solium are from nine to twelve in number, and of the T. saginata fifteen to twenty, and the latter are much smaller.

Treatment.—There are two separate stages in the process of expulsion of the parasite—the preparatory treatment; the exhibition of the tæniafuge. The preparation of the patient consists in the use of a laxative to remove mucus and other matters in which the *scolex*, or head, is imbedded, and to prevent accumulation of such matters by a low diet, which will leave almost no residuum. Sulphate of magnesia should be administered each morning for two mornings before giving the remedy—one or two teaspoonfuls at a time in sufficient water. The diet should consist of milk, steak, tea, and toast, for the day before and during the treatment. German practitioners cause the patient to take certain articles which experience has shown are highly disagreeable to the parasite—such as garlic, onions, and salt-herring—and accordingly they direct a plateful of herring-salad, a savory dish made up of those articles, agreeable enough to Germans, but highly distasteful to tape-worms! The medicine need not be given on an empty stomach; the patient may take a cup of coffee before beginning the medicine. Many remedies have been proposed, and opinions are diverse as to their utility. Heller prefers kousso; Cobbold,[*] extract of male fern; while Davaine does not indicate his preference; and Küchenmeister,[†] after an exhaustive examination of the almost innumerable methods, ancient and modern, declares his preference for the decoction of pomegranate. The author's experience, which has been not inconsiderable, is decidedly in favor of the pomegranate. The most successful treatment of tape-worm the author has any knowledge of, is that of an ignorant barber, who has a secret method which seems never to fail. He does not attempt any preparatory treatment, but administers his medicine (apparently, a decoction of pomegranate) in the morning, the patient fasting, and retires from the house with the worm and his fee in the afternoon.

Küchenmeister prepares his decoction of pomegranate as follows: ℥ iij of fresh bark, after macerating for twelve hours in ℥ xij of water, are concentrated to ℥ vj by a gentle heat, and this fluid is taken in three doses within an hour. He precedes the administration of the pomegranate by one day of fasting, and ℥ ij of castor-oil, taken the night before. He prefers to add to the pomegranate the ethereal extract of filix mas and extract of tansy, ℈ j — ʒ ss of the former and ʒ ij of the latter.[‡]

Heller administers the kousso in a special manner—by the method of Rosenthal—which consists of compressed balls or disks coated with gelatine. Five drachms is the quantity required for a T. solium, and seven and a half drachms for a T. saginata. The gelatine-coated balls and disks are placed as far back on the tongue as possible and swallowed alone, or aided by some coffee. The tendency to vomit must be resisted—mustard applied to the epigastrium, small bits of ice swallowed, the recumbent posture maintained. Two hours after the last bolus, an ounce or two of castor-oil should be administered, the object being to expel the worm speedily and entire. Heller affirms that this method is highly successful, but Küchenmeister thinks kousso an uncertain remedy. The author's experience with it has been unfavorable—it expelled a large quantity of the worm, the segments, but not the head or scolex; but it was adminis-

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[*] "Entozoa," *op. cit.*, p. 233.

[†] "On Animal and Vegetable Parasites of the Human Body." By Dr. Frederick Küchenmeister. Sydenham Society edition, vol. i, p. 171.

[‡] The active principle of pomegranate—*pelleterine*—is now generally preferred. In a communication to the "Bull. Gén. de Thérap.," July 15, 1879, Dr. Bérenger Ferand reports comparative trials with the tannate and sulphate of pelleterine, prepared by M. Ch. Tanret, the discoverer. He finds the tannate more efficient. The dose is from five to fifteen grains, administered fasting, the diet the previous day consisting of milk and bread. The remedy is followed by compound tincture of jalap, or castor-oil, or sulphate of magnesia. Tanret has put on the market, as a proprietary medicament, a concentrated fluid extract, which is sold under his name. The author has found it to be an efficient remedy.

tered in a decoction, the patient swallowing a great mass of leaves, stems, and flowers, so that vomiting could hardly be resisted. The method by fern consists in the administration of the so-called ethereal extract—the oleoresin—in ʒ ss doses, fasting. It is most pleasantly taken in perles or capsules. If of good quality, and given after suitable preparation in an efficient dose, it is a successful remedy—according to Cobbold, the best of the group of tæniafuges. The seeds of the common field pumpkin is a homely but very efficient remedy, which deserves to rank among the best of the class. The fresh seeds are rubbed up into an emulsion by the addition of some water, the woody fiber separated by a coarse sieve, and the mixture drunk fasting. Usually no purgative is required, but one should be given if the bowels do not act promptly. The failures are due, simply, to the difficulty of retaining a sufficient quantity. A great many cures have been effected by turpentine; it is, indeed, one of the most efficient of tæniafuges, but the natural repugnance to swallowing such a dose, the powerful effects produced by it, and the subsequent ill results, are such as to hinder its employment, and to restrict it to the cases which have resisted other means. Large doses, acting promptly as a cathartic, are not so injurious as the smaller doses which pass off by the kidneys. From one to two ounces of turpentine and as much castor-oil are administered together. Pelleterine has been used with increasing success since its discovery, but considerably larger doses are required than were at first supposed to be necessary. Preparation of the patient is not essential. Recently chloroform has come into use and is warmly commended, numerous successful cases having been reported. It is given in from ʒ ss to ʒ ij, mixed with twice its quantity of glycerin. It may be inclosed in capsules, or given in an emulsion. The former plan is probably more effective. The stools should be carefully and minutely inspected, for the medicine is not successful if the scolex is not expelled. The head with its row of hooklets, its suckers, etc., can be recognized by the naked eye, but an ordinary pocket lens will bring out all parts with sufficient distinctness to render an inspection positive. If the scolex is not found, and is retained, in six weeks to three months the segments or proglottides will be passing again.

Bothriocephalus latus is usually classed with tape-worms, and clinically properly so, but, zoölogically considered, it is not a tape-worm. Its *habitat* is the small intestine—its scolex attached to the mucous membrane of the duodenum by its suckers. It is found more frequently in the adult and in the female. Its size is greater than that of tænia; its segments are not detached at maturity, and do not maintain an independent life. Detached parts of considerable extent are expelled at long intervals. It is ordinarily, but not invariably, soli-

NOTE.—A combination of the most active tæniafuges, consisting of pomegranate, fern, koussin, etc., is prepared by several pharmacists—among them Wyeth & Brother, of Philadelphia—and sold as a proprietary medicine.

tary. According to Odier, who has observed many cases at Geneva, the bothriocephalous causes swellings of different parts of the abdomen, irregular stools, nausea, vertigo, palpitations, night terrors, etc. There may be no symptoms at all, and, when symptoms do occur, are about the same as those already described for tænia. The expulsion of the bothriocephalus is accomplished more readily than is the tape-worm. Kousso rarely fails. The oleoresin of filix mas is also successful. Kameela has been found efficient. In fact, any of the remedies already referred to as tæniafuges may be used against this worm. In Switzerland, the secret remedy of Peschier, supposed to be fern, is much used.

NEMATODA—ASCARIS LUMBRICOIDES—ROUND WORMS.

General Considerations.—The lumbrici are found under all conditions of climate—in cold, in warm, in moist, and in dry climates. They sometimes appear so generally as to become epidemic. In certain epidemics of dysentery, worms in large numbers appeared in the evacuations. But these observations, made in the last century,* are open to suspicion, for in those times the pathological importance of worms was much greater than now. It is true, even now, under certain local conditions, that worms are very common—so much so as to constitute an epidemic, and, in some epidemics of fever and of dysentery, great numbers of worms appear in the intestinal tract. The chief mode of propagation is by drinking-water. The ova of the round worm resist freezing and a very high temperature, and are surrounded by such a strong envelope as to oppose successfully ordinary destructive influences, and live for years. It follows that, in country places, where human excreta easily gain access to drinking-water, numbers of people may be simultaneously affected, or in quick succession. Filthy habits of a people—of a community of negroes, for example—contribute greatly to the propagation of lumbrici, by the dissemination of ova through articles of food and drink.

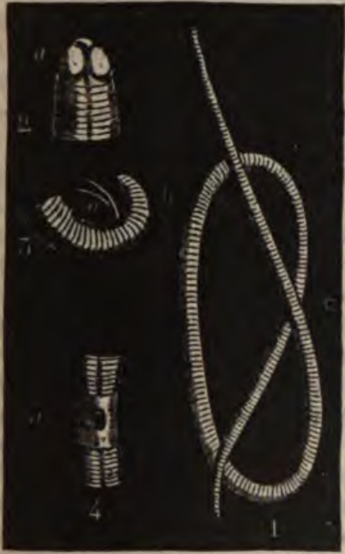

Fig. 10.—Ascaris lumbricoides —1, complete worm; 2, head; 3, tail of the male; 4, middle of the body of female.

The number of ascarides existing at one time in the intestinal canal is various: there may be one, two, or three worms, or they may reach five hundred or thousands. When very numerous, they may be grouped in rolls or bundles, distending the whole or a part of the intestine, or occluding it. Their place of sojourn is in the small intestine. They occur in early life chiefly, although Heller asserts the contrary, and are not common under one year and after twenty. Females are more subject to them than males, and feeble, lymphatic, and strumous persons more than the robust. Poor aliment, a vegetable diet, and fermented drinks favor their development. Autumn is the season of their greatest prevalence. From their origin to the end of their existence rarely does more than a year transpire, but our knowledge on this point is not very definite.

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* Davaine, *op. cit.*

Development.—The lumbricoid worm (Fig. 10) is cylindrical in shape, reddish-brown or brownish-yellow in color, and tapers at both extremities; but the cephalic extremity is larger, and contains at its summit three lips or papillæ, having the mouth between them. The male is smaller than the female, and is distinguished by the tail being always turned toward the abdomen like a hook. The ova, which exist in almost incredible numbers, are oval in shape, have an extremely tough, double shell, and dark, granular contents. The eggs when expelled are slow to develop, several months, sometimes years, being required. "They do not lose their power of development for several years, and the young embryo, while in the shell, also retains its vitality for years." The subsequent steps in the development of lumbrici are at present quite unknown.

Symptoms.—When few in number, as is the rule, the host being in good health, there are no symptoms of any kind produced by them. When very numerous, disorders of digestion, of nutrition, and of the nervous system, are caused; but these results are not peculiar to the round worm, and have been alluded to in connection with the tapeworm. The usual symptoms are colicky pains about the umbilicus; tumefaction of the abdomen; capricious appetite, now insatiable, now wanting; occasional nausea and vomiting; sometimes diarrhœa and stools containing mucus mixed with blood; whey-like urine; itching of the nose and anus; bluish coloration of the lower eyelid, dilatation and sometimes inequality of the pupils; emaciation; irregularity of the pulse; choreic and hysterical seizures; restless nights, terrors, and grinding of the teeth in sleep, etc. No confidence can be placed on the diagnosis of worms when all of the foregoing symptoms are present, for they are much more frequently produced by other causes. Hence, the diagnosis must be largely conjectural unless worms are passed from time to time. One or more may be found in the stools, and not rarely worms are brought up from the stomach, and excite gagging and strangling until disengaged from the fauces. If the symptoms above mentioned persist after the ocular demonstration of the presence of worms, they are probably due to this cause. Chorea and epileptiform attacks, in girls of eight to fifteen, may be due to the presence of worms, and cease on their removal—of which numerous examples have fallen under the author's observation. Occasionally obstruction of the intestine has been caused by a bundle of worms—either within the abdomen, or in a herniary protrusion. Requin narrates a case, the obstruction occurring at two points—in the small intestine; at the middle of the transverse colon.

Ascarides crawl up into the pharynx, the Eustachian tube, the nares, and the larynx. Aronssohn has collected several cases, Davaine others, of death happening suddenly with symptoms of suffocation due to worms crawling into the larynx. Thirty-seven cases are reported (Davaine) of lumbrici in the biliary passages, in the substance of the liver, or in the cavity from rupture of the duct. The most usual position for them is the common duct, which they obstruct, jaundice results, and ultimately serious derangement of the liver ensues. Hepatic abscess is also a result, but, very rarely, of the lodgment of a worm which has passed up into the body of the liver, and excited suppurative inflammation. In some rare cases a worm has been discharged by an hepatic abscess opening externally. Worms have also been discharged externally by fecal abscesses, and they not unfrequently pass into the cavity of the peritoneum through perforations of the intestines. Abscess of the pancreas has been caused by a round worm blocking the duct, an example of which has been reported by Dr. John Shea.

Treatment.—There are various remedies highly effective in the removal of the ascaris lumbricoides. The most generally used is *santonine*, or santonic acid, the active constituent of artemisia santonica. The advantage of this, besides its efficiency, is the slight taste and ease of administration. It should always be explained that the vision of those taking santonine is affected: all objects seem as if looked at through yellow-colored glasses, and also that the urine is stained a deep yellow. In overdoses santonine causes violent nervous symptoms. It is given in the form of powder, rubbed up with sugar, or some extract of liquorice—two to four grains at night, followed by a laxative in the morning. Calomel has considerable vermifuge property, and is often alone sufficient, but is now used as an adjunct to santonine, two to four grains given with the same quantity of santonine. This plan, which is very satisfactory, is still more efficient if the use of the vermifuge is preceded by hydrocyanic acid (the officinal dilution), two or three drops, three times a day, for two days. Next to santonine in point of efficiency is chenopodium or worm-seed, which is usually administered in the form of the oil. Its powerful odor and disagreeable taste are strong objections. Five to ten drops can be given in an ounce of castor-oil, or in the fluid extract of spigelia, also an efficient vermifuge. The fluid extract of spigelia (pink-root) may be given alone in from one to four drachms at a dose, or in the officinal combination, the fluid extract of senna and spigelia. Any of the remedies named are efficient against the round worm.

OXYURUS VERMICULARIS.—THREAD-WORM.

Description.—This parasite (Fig. 12) derives its common name—thread-worm—from its whitish appearance and size, like a bit of fine sewing-cotton, and from its habitat, the seat-worm. There are two sexes, male and female, the male being only one half the size of the female. The female worm is scarcely a half inch (nine to twelve mm.), and the male is about one fourth of an inch (three to five mm.) in length, cylindrical, tapering to both extremities, but the cephalic end is blunter. The ova are contained in a stout envelope which resists considerable heat as well as cold, but softens in the intestinal canal of man, and discharges its embryo, which, indeed, may be discerned in the mature eggs already in process of development. The habitat of the oxyurus is the large intestine of man, especially the rectum, and they insinuate themselves into the folds of the mucous membrane and skin at the margin of the anus. They are most abundant in early life, and sometimes at the other extreme, in old age.

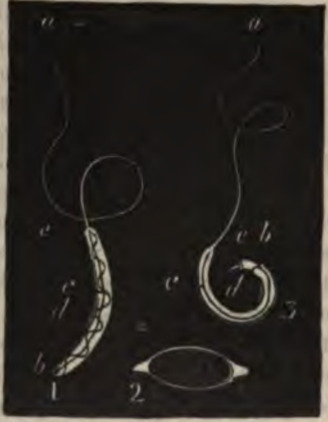

FIG. 11.—Trichocephalus of Man.—1, female: *a*, cephalic extremity; *b*, caudal extremity and anus; *c*, *d*, digestive tube and ovary; *e*, orifice of sexual apparatus. 2, isolated egg. 3, male: *a*, cephalic extremity; *b*, anus; *c*, digestive tube; *d*, spicula or penis; *e*, sheath into which it is withdrawn.

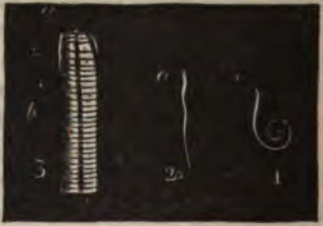

FIG. 12.—Oxyurus Vermicularis.—1, male of natural size; 2, female, ib.; 3, cephalic extremity, magnified.

Symptoms.—They excite by their presence in the rectum an intolerable itching, sometimes severe pain, tenesmus usually, and these sensations are propagated to the genito-urinary organs. The tormenting itching occurs at special times, and is very aggravating at night, when warm in bed. The stools are usually a little relaxed, fetid, and coated with mucus, and occasionally streaked with blood. An inspection of the parts discloses a reddened and roughened integument all about the anus, and excoriations of the mucous membrane caused by the repeated friction of the parts. The worms may often be seen *in situ*, or in the evacuations, but it is necessary sometimes to administer an injection or a laxative to procure ocular evidences of the presence of these parasites. Besides the local, various reflex phenomena are induced by the irritation of the oxyurus, as epilepsy, chorea, catalepsy, etc. Unquestionably, excitation of the sexual organs is thus caused, leading to onanism. Besides the reflex, direct irritation of the genitals in girls is set up by the presence of these worms in the vagina, where they deposit their ova and develop in immense numbers. Violent local inflammation and a blenorrhagic discharge are also induced in this way, exciting suspicion of gonorrhœal infection. The oxyurus is not confined to the rectum, nor are its excursions limited to the perineum and vagina. It migrates upward into the large intestine, develops in the cæcum, and the lower part of the ileum is also invaded. So that, although the proper habitat of the parasite is the rectum, it should not be overlooked that they exist in the cæcum and in the lower part of the ilium in great numbers.

Treatment.—The fact just stated in regard to the position of these parasites in the intestinal canal is of great importance in the treatment. The administration of one of the vermifuges, especially santonine, aided by calomel, should be the first step in the treatment. As soon as this has acted, the bowel should be irrigated by a weak decoction of quassia or of aloes. A simple injection will usually suffice, since the santonine has probably displaced all of the parasites above. The decoction should also be used as a vaginal injection, employing a very small tube, so that all of the canal can be reached. As the ova are deposited in the folds of the anus, and are not reached by the injections, the next step consists in carefully sponging out all the folds and crevices of the anus and perineum, and the external genitals also, with a one per cent. solution of carbolic acid. If treated in this thorough manner, the applications being repeated a few times, the parasites will be entirely destroyed, but neglect of any of these precautions will render repeated applications necessary. Solutions of carbolic acid as an injection have been used with success in the treatment of the oxyurus, but such serious symptoms have arisen in some cases that this practice ought not to be continued.

Trichocephalus (Fig. 11) is rarely encountered. In respect to clinical history and symptoms, it does not differ from the round worm.

DISEASES OF THE PERITONEUM.

PERITONITIS.—INFLAMMATION OF THE PERITONEUM.

Definition.—Inflammation of the peritoneum occurs in two forms—*acute* and *chronic*. It may be limited to a part, or involve the whole of the membrane: in the former it is *local*, in the latter *general* peritonitis. It may be an independent affection, or *primary*, or it may be caused by the extension of a morbid process, from adjacent organs or tissues, or *secondary*.

Causes.—As a primary disease peritonitis is rare, but it may occur at any age, even during intra-uterine life. Intense cold, severe and protracted counter-irritation by blisters, and blows on the abdomen, may excite the inflammatory process. Very much the most frequent cause is the extension of internal lesions of the abdomen—e. g., perforations of the stomach, intestines, bladder, etc., or inflammation of these organs. To this category may be added the causes of pelvic inflammation of the uterus and annexed organs. It is not unfrequently an intercurrent malady coming on in the course of certain cachexiæ, as pyæmia, albuminuria, and the eruptive fevers.

Pathological Anatomy.—The first step in the inflammatory process is the occurrence of hyperæmia, the capillaries being enlarged and distended, and the blood-pressure is so increased within the area of inflammation that extravasations of blood occur at various points. An arrest of the normal secretion and an abnormal dryness are then evident; next an exudation, very thin but adhesive, forms on the inflamed surface and glues the neighboring parts together, but not firmly, for they may be easily separated. Simultaneously, a reddish, serous fluid is poured out into the cavity. The inflammation will now assume one of two directions—it will take the *adhesive* or *exudative* form. The fibrinous exudation already mentioned is almost pure fibrin and contains but few cellular elements. Presently, however, the cells of the endothelium become swollen, their contents granular, and their nuclei undergo multiplication. If, now, the process ends with the adhesive inflammation, the proliferation of the endothelium will soon be arrested, a delicate connective tissue will be formed from the new cellular elements, blood-vessels soon appear, and a distinct neo-membrane is the result, binding neighboring surfaces together, or forming bands of adhesion of greater or less extent. If the inflammatory process assumes the other direction, the effusion increases. It is at first sero-fibrinous, i. e., a serous fluid, having masses of flocculi of lymph floating in it. The deposit of fibrin, which in the other form (adhesive) is slight in extent, and which disappears in the process of formation of the neo-membrane from the new cells, in this form (exudative) is very much increased, and constitutes a coating of considerable thickness. The endothelium undergoes extensive proliferation; the connective-tissue corpuscles of the basement membrane also, and new vessels develop. On separation of the fibrin layer from the serous membrane, the latter bleeds from rupture of minute new-formed vessels; it appears dense, thick, and œdematous. The swelling, hyperæmia, and œdema, also extend to the sub-peritoneal connective tissue, and ultimately to the muscular tissue, which in turn becomes softened, pale, and flabby. When the inflammation occurs in the peritoneal layer of the liver or spleen, the tissue adjacent to the inflamed membrane is paler than normal, softened from œdematous infiltration, and otherwise altered. The effusion poured out into the cavity assumes various appearances and characteristics. The quantity varies from a few ounces, in the dependent parts of the cavity, up to several gallons. It may be sufficient to force up the diaphragm to a level with the third rib, make the heart lie transversely by pushing up the apex, displace the lungs, etc. The effusion may be chiefly fibrinous with but little fluid. When this is the case, the thickest deposits are seen over the solid organs, the liver and spleen, and it may be general, uniting the whole surface, or limited in extent, forming occasional adhesions. The neo-membrane contains vessels, often of considerable size, and having walls of exceeding tenuity. These vessels rupture easily, and considerable hæmorrhage results, and this, mixed with the effusion, constitutes another form, the so-called hæmorrhagic effusion. The adhesions, when isolated and not general, undergo great changes ultimately, by reason of the extensive motion possessed by the abdominal organs. They may, by subsequent contraction, cause great deformity of organs and seriously impair their functions, and in the case of the intestine may induce twisting, encroach on their caliber, and bring about slow occlusion. The small intestines may by means of such adhesions be agglutinated together, forming an almost solid mass, irregularly rounded, as the author has seen, in certainly one well-marked case. The effusion may be serous—a faint greenish, or greenish-yellow, or milky fluid, similar to the fluid of ascites, except in the presence of flocculi of fibrin, bits of false membrane, and casts of cells of the endothelium. The effusion is sero-fibrinous, when there is a large quantity of fibrin suspended in it. When absorption of the fluid takes place, the solid exudation undergoes the changes already described. The effusion may be purulent. When this is the product of the inflammation, its cause is, as a rule, perforation and the escape of purulent or decomposing matters into the peritoneal cavity. When the effusion is purulent, the amount of fluid contained in the abdomen varies greatly. There may be thick masses of pus, or the pus may be mixed with a quantity of serum, constituting a sero-purulent fluid.

The changes of chronic peritonitis are similar to those of the acute form. There is often little or no fluid exudation, and when present is not abundant, and has a purulent or sero-purulent form. The principal fact is the existence of false membrane, either general or in local bands. The intestines, as already described, are sometimes united in a bundle and form a globular mass of some compactness. Occasionally a part of the neo-membrane, especially where it has attained the greatest thickness, undergoes a calcareous transformation; or it may become soft, friable, and granular, doubtless preparatory to absorption, or it may be converted into connective tissue. Divided by membranous adhesions, the cavity of the peritoneum may be converted into various secondary cavities, some containing serous and others purulent collections. The latter may be converted ultimately into a cheesy mass. In chronic peritonitis, tubercular deposit is common, and gray granulations are disseminated through the false membrane and the sub-serous connective tissue. Tuberculous peritonitis is usually connected with tuberculous ulceration of the mucous membrane of the intestine, and tubercular adenitis of the mesentery, and is coincident with pulmonary tuberculosis.

Symptoms.—When idiopathic or primary peritonitis occurs in a previously healthy individual, it sets in with a chill, an intense fever, and very severe local pain and tenderness. If it succeeds to a perforation, the onset of the peritoneal mischief is announced by an intense pain, felt in the region of the accident, and rapidly extending thence over the abdomen. Then the fever movement is but slight. If peritonitis from perforation happens in the course of typhoid fever, or in any other adynamic state, there may be few symptoms besides distention of the abdomen and increase of the adynamia. When it results from an extension of inflammation by contiguity of tissue, it is announced by an exaggeration of the fever, by pain and tenderness of the abdomen, and by vomiting—the last-named symptom being especially significant if it has not existed in the case previously. In what mode soever peritonitis may begin, the symptoms most characteristic are, pains in the abdomen, gaseous distention, rapid failure of strength, and fever, somewhat remittent in type, with the remission in the morning. The pain in the abdomen is usually an intense, cutting, boring pain, somewhat more severe at certain places, but felt all over the abdomen. The slightest touch aggravates the pain, and hence the patient avoids movement, suppresses cough, and breathes with the chest-muscles. For the same reason the breathing is short, quick, and superficial, to avoid motion of the diaphragm. The decubitus of the patient is unconsciously assumed to prevent pressure of the muscles on the tender peritoneum. He lies on his back, if the peritonitis is general, with the thighs flexed on the pelvis and the shoulders elevated, and, when he is told to extend the limbs, he does so very cautiously and soon abandons the attempt, his countenance as well as his expressions indicating the increased pain the effort has given him. In the beginning of the disease, the abdominal muscles are kept contracted and rigid to guard the peritoneum from injury by movement, but it is also a reflex state of tonic muscular contraction, which occurs simultaneously in the muscular layer of the bowel, and is due to the irritation of the terminal nerve-filaments in the peritoneum. But paresis of the bowel soon succeeds to tonic rigidity, in accordance with another law—overstimulation, or long-continued, exhausts the irritability of the organic muscular fiber. The bowel then becomes extended by the accumulating gas, and soon (on the second or third day) an extreme degree of meteorism is the result, which, in fatal cases, continues up to death. This extreme distention of the abdomen adds to the difficulty and pain of breathing. The sonority of the percussion-note is tympanitic over the course of the large intestine especially, and the abdomen generally, except the dependent parts in the flanks and iliac fossæ, where the accumulation of fluid imparts to it the character of dullness. The normal hepatic dullness lessens materially or disappears, because of the displacement of the liver upward and its partial rotation on its long axis. The position of the dullness on percussion varies with the changes of position of the patient. It is occasionally possible to hear a friction-sound by auscultation, but the duration of it in any case is very brief. The tongue is coated and the appetite impaired at the onset. Rarely is vomiting absent. It begins soon after the disease sets in, and at first articles of food and gastric mucus come up, then biliary matters from the duodenum. Vomiting may occur spontaneously, or be excited by taking medicine, food, or drink. In some rare cases the vomiting has been incessant, and finally stercoraceous. In such cases obstruction is supposed to exist, but not confirmed on *post-mortem* examination, only peritonitis being found. Constipation is the rule in case of peritonitis, but occasionally diarrhœa is present; then, usually, some coincident disease of the bowel exists, as tuberculosis or septicæmia, for example. Constipation is the necessary result of the paresis of the bowel; but paralysis of the sphincter may be so complete as to permit the escape of fecal matters by mere pressure on the abdomen. An extension of inflammation to the vesical peritoneum causes strangury and irritable bladder. Hiccough is a frequent and most distressing symptom, and is due to a reflex irritation of the diaphragm, transmitted from the nerve-endings in the peritoneum. The pulse in peritonitis is small, quick, and frequent, the tension high, and when cardiac failure comes on in fatal cases it becomes excessively quick and small, and may disappear at the wrist when the heart is still acting. It will range in ordinary cases from 100 to 140; when collapse approaches, the pulsations may reach 160 to 200. When collapse comes on, the temperature, which had risen to 103° Fahr., sinks below normal. As has been already pointed out, the respirations are costal in type, very shallow, and becoming more so with the failure of the vital powers. There is then cyanosis. The countenance is anxious, shrinks; dark, livid circles surround the eyes. In collapse the surface is cold, wet with a cold sweat, the skin wrinkled and sodden, the body exhales a cadaveric odor, the voice is husky, but the mind remains clear though rather apathetic, and at the last the brain is clouded by carbonic-acid poisoning. Or, instead of an unclouded intellect, there may be delirium from œdema of the brain, and, extremely rarely, unconsciousness soon after the onset of symptoms. In many cases, as collapse develops, the peculiar type of respiration—the Cheyne-Stokes respiration—appears, and is highly significant of a fatal termination.

Course, Duration, and Termination.—The course of peritonitis is rapid, the mortality great. The usual termination is in death. When it arises from perforation, a fatal result may occur in two or three days, and, when it is idiopathic, in five or six; but the cases of this variety may last two to three weeks. Peritonitis due to internal obstructions adds to the severity of the symptoms and the gravity of the case, but its course, apart from the principal malady, is not well defined. The gravest cases are those which occur in the course of septic diseases, or are due to the escape of decomposing and irritating matters, by a perforation into the cavity. The only forms which may be regarded as at all favorable are those due to the extension of a simple inflammation, by contiguity of tissue, from the abdominal or pelvic viscera. In these the inflammation is simply exudative and adhesive, or sero-fibrinous. When improvement begins, it is announced by a diminution of the pain, lessening of the meteorism, and cessation of the vomiting. A case of acute peritonitis may terminate in a chronic form of the disease. After a period of improvement, grave symptoms will again set in, induced by the changes in shape, position, and functions of organs, the result of adhesions, contractions of bands of lymph, etc.

Prognosis.—The statements already made sufficiently set forth the grave character of peritonitis. The prognosis in the mildest cases must be guarded, and in all severe cases unfavorable.

Diagnosis.—Peritonitis is to be differentiated from hysterical tenderness of the abdomen, rheumatism of the abdominal muscles, and acute painful affections of the various organs. From hysteria it is differentiated by the hysterical history, by the crying, sobbing, and globus hystericus, by the absence of all constitutional symptoms, and finally by the tenderness being merely an hysterical condition, excessive on the surface, but permitting, when the attention is withdrawn, firm, deep pressure. The suffering of the hysterical state differs from real pain in the disproportion of the expressions and the evidences; while the most extravagant terms are used to describe the pain, the countenance is placid. In rheumatism of the abdominal muscle, there will probably have been other cases of the rheumatismal character; the pain is limited to the muscles, and deep pressure does not increase it, and the constitutional state does not indicate a severe disease. In acute painful affections it is sometimes difficult at once to decide, but as a rule these begin rather more abruptly, the pain is more acute, and there is not usually a history of a disease from which peritonitis might be expected to arise. The great majority of cases of peritonitis arise from previous disease in the peritoneal or pelvic cavities; it is extremely rare, indeed, for an idiopathic case to occur.

CHRONIC PERITONITIS.—There are two forms: 1. Succeeding to the acute; 2. Tubercular. The acute symptoms subside and there is a gradual absorption of the fluid portion of the exudation. A sero-fibrinous exudation may undergo conversion into a purulent; the fever, which had diminished or ceased, rises again and takes on the septicæmic character—there are chills, fever, and sweats. Rapid decline of the vital powers takes place under these circumstances. Or the effusion may become encysted by the formation of adhesions, as already described, and become a pus-depot, which may be converted, ultimately, into a caseous or calcareous mass. In other cases these purulent collections behave as ordinary abscesses, and manifest a tendency to find their way externally. Abscesses formed above a line drawn transversely across the abdomen through the umbilicus tend to dissect upward, and make their way out through the lungs; those below this line tend to pass down along the course of the femoral vessels. Although there are many exceptions, this may be considered as a natural tendency. In the dissections made by these abscesses, fistulæ may be established externally, with different parts of the bowel, with the thoracic cavity, etc.; or rupture may occur into the peritoneal cavity, again exciting fresh inflammation. The chronic, local, and partial peritonitis, about certain organs, may set up important changes by the metamorphoses of the exudation. Thus, thick and contracting connective tissue about the gall-bladder, and on the upper surface of the liver, compresses the organ, or may obstruct the hepatic duct or the portal vein. The tubercular form of chronic peritonitis is often associated with the corresponding disease of the lungs, or intestinal mucous membrane, or of both. Its onset is obscure, and development slow, so that weeks or even months may pass before the patient is so reduced as to take to his bed. It usually sets in by colicky pains felt especially during the time digestion is going on. Constipation alternates with diarrhœa, and there may be, but not invariably, attacks of vomiting, the matters thrown up consisting of mucus and greenish, bilious-looking matter. The attacks of vomiting may coincide with the colic-like pains.

The patient rapidly declines in flesh and strength. There are daily chilliness and febrile movement. The skin is harsh and dry ; sweating usually occurs at night ; the urine is scanty, high colored, and deposits an abundant uric-acid sediment. With the development of these symptoms the abdomen gradually assumes a characteristic condition. By the accumulation of gas in the intestine, and of serous effusion in the cavity, the abdomen enlarges. Notwithstanding a considerable effusion, it is rare that the signs and symptoms of ascites are present. There is dullness in the dependent parts, whatever may be the decubitus of the patient, but not such a fluctuation as occurs in ascites. The compression of the vessels, by the effusion within and the direct pressure of membranous adhesions, but especially the matting of the small intestines into a globular mass, and the pressure of this tumor-like body on the iliac veins, cause an extensive œdema of the lower extremities, the scrotum, and the abdominal walls. This result is promoted by the enlargement of the mesenteric glands, which are also occupied by tubercular deposit. The course of this malady is slow, but the termination by death is not less certain. The reader should not overlook the distinction between a tubercular peritonitis occurring with tubercular phthisis and other tubercular diseases and a peritonitis in which tubercular deposit is secondary to the morbid process which had preceded it.

Treatment.—When robust subjects are attacked by peritonitis, there can be no doubt of the utility of leeches, ten to twenty applied over the abdomen. In the cases of local peritonitis (typhlitis, for example), if the patient is not very weak, leeches are highly serviceable. There are few, indeed, who can not bear the loss of blood by two or three leeches. The time for their application is at the onset of the disease, before solid exudations have occurred. After leeches, or at once, an ice-bag should be applied to the abdomen, or to the part only affected. This ceases to be useful, and is better supplanted by warm applications, when exudations take place and the abdomen swells. With the first symptoms, morphine should be administered hypodermatically, and should be repeated every four, six, or eight hours according to the effect, such a degree of narcotism being maintained that pain is relieved, the pulse considerably reduced, but yet the patient is easily roused. Atropine should be given with the morphine. The very heroic use of morphine, advocated in some quarters, is not to be commended. The best curative results are obtained from doses that affect decidedly without inducing a degree of narcotism that may be dangerous. At the very beginning, the administration of antipyretic doses of quinine is in a high degree beneficial, and the effect may be maintained by frequent exhibition of smaller doses. This ceases to be useful when there is solid and liquid exudation. When effusion occurs, another and a very different kind of medication must be adopted. The decline of the vital powers must be retarded by suitable nutrients and stimulants. The local applications should consist of warm fomentations, mustard-plasters, or flying-blisters, or the tincture of iodine. By the stomach the salts of ammonia should be administered freely, and morphine continued *pro re nata.* Ten grains of the carbonate of ammonium, in an ounce of the solution of the acetate, every four hours, when the exudation is going on, is, the author believes, a remedy of the highest utility. In the peritonitis from perforation, absolute repose, opium, ice, and the avoidance of all foods and drinks, are the proper measures.

ASCITES—DROPSY OF THE ABDOMEN.

Causes.—The chief factor in the pathogeny of ascites is mechanical obstruction of the vessels, the portal system, and the most common cause of this obstruction is cirrhosis of the liver. Tumors, as aneurism of the hepatic artery, tubercle masses, cancer, and hydatids, in a situation to compress the portal vein, will also cause an effusion into the peritoneal cavity. Increase of pressure in the portal system may be due to obstructive disease of the heart or lungs. Again, dropsy of the peritoneum may be part of a general dropsy, especially in chronic nephritis. Accumulation of fluid is a result of peritonitis, acute or chronic, but this does not, properly, constitute ascites.

Pathological Anatomy.—The amount of effusion which exists in ascites varies from a few ounces to many gallons. It is usually of a pale straw-color, or it may have a greenish tint, and is transparent, and may be free from flocculi, or any foreign constituents. Its reaction is alkaline, and its specific gravity below that of the serum of the blood. It contains albumen or albuminate of soda, but the proportion is less than is present in blood-serum, but greater than in other serous exudation except hydrothorax. The biliary acids and pigment are also found in the ascitic fluid, when jaundice exists, and creatine and creatinine are very common constituents. In many cases fibrin is held in solution, and slowly coagulates in an exceedingly fine reticulation of fibers. Sometimes ascitic fluid is reddish from the presence of blood derived from ruptured capillaries; again, blood may indicate the probability of cancer. The peritoneum long in contact with fluid is altered in character and appearance by imbibition ; it becomes sodden, cloudy, and thickened, but these are not inflammatory changes. The distention of the cavity and the displacement of organs disturb the relation of the parts.

Symptoms.—As a rule the beginning of ascites is obscure, and it is not discovered until the sense of fullness and tension directs attention to the part, or an examination of the abdomen is made for the purpose, existing lesions rendering it probable that effusion has occurred. An increasing fullness of the abdomen is the most important objective symptom. It is not wholly fluid, but the distention is in part due to flatus in the intestines and fecal accumulations, the result of constipation caused by pressure on the sigmoid flexure. If the patient is erect, the fluid distends the iliac and hypogastric regions; if lying down, the fluid flows to the sides; if turned upon one side, the fluid takes a corresponding position—so that the dullness on percussion varies with the posture of the patient. With the increase in the amount of fluid the girth of the abdomen enlarges, so that in cases of large effusion the abdomen may be two or three times larger than the normal. When the effusion is great and of long standing, the umbilicus is forced outwardly, and forms a tumor with thin walls, and soft and fluctuating in character. The physical signs are characteristic: On mensuration, the increased circumference; on palpation, a peculiar wave-impulse communicated through the intervening fluid, when a slight blow is made on one side; on percussion, a tympanitic note over the distended bowel, and a region of perfect dullness corresponding to the position of the fluid. The wave of fluctuation is best felt by laying the hand extended flat on one side of the abdomen, and gently tapping the opposite side. The distended abdomen forces the diaphragm upward and therefore embarrasses the respiration and the cardiac movements; the urinary secretion is diminished because of the pressure on the renal arteries and veins, and of the escape of fluid into the peritoneal cavity; constipation results from the compression of the sigmoid flexure. The integument of the abdomen has a glistening appearance, arising from stretching and œdema, but the skin generally is harsh and dry. The lower extremities and the scrotum also are much swollen, when the ascitic fluid is sufficient in weight to compress the vena cava and iliacs.

Course, Duration, and Termination.—The course and behavior of ascites depend much on the cause producing it. Usually the effusion occurs slowly, as, for example, in cirrhosis, in which disease there may be months occupied in producing sufficient effusion to distend the abdomen. In idiopathic ascites, the accumulation may take place in one or two weeks. The amount of increase in the blood-pressure may vary greatly when an obstruction, cardiac, pulmonary, or hepatic, is the cause of the effusion. Idiopathic ascites is shorter in duration than the other forms, and terminates in health in a few weeks. The duration of the other forms is a question of the course and behavior of the malady, of which ascites is usually a symptom. When dependent on obstructive disease of the heart, lungs, or liver, especially the liver, the duration is indefinite. The fluid may be removed by treatment, and return again and again, for the original cause remains.

Prognosis.—The question of recovery is determined by the presence or absence of certain organic changes. If the effusion is simply peritoneal, the prognosis may be favorable. If it is a symptom of cardiac, pulmonary, or hepatic disease, the prognosis is unfavorable, for these maladies being incurable the effusion will recur, if at any time it may be removed.

Diagnosis.—Ascites must be differentiated from ovarian tumors, pregnancy, distended bladder, chronic peritonitis, and enlarged spleen. As ovarian tumors are so often accompanied by effusion into the peritoneal cavity, mistakes are frequent, ovarian tumors being confounded with ascites, and *vice versa.* The distinction lies in the following considerations: Ascites is almost always preceded by obstructive diseases of the heart, lungs, or liver, especially by cirrhosis, and the derangements of health which the existence of these obstructive diseases always implies. Ovarian disease does not necessarily impair the health, and is not preceded or accompanied by the lesions pertaining to ascites.

In ascites the enlargement of the abdomen is uniform, begins at the dependent part, whatever that may be, and the dullness on percussion changes with the position of the patient; ovarian tumor begins in the iliac fossa of either side, the growth is obliquely upward, does not change its position according to the posture of the patient, nor does the dullness change. The tympanitic percussion-note, derived from percussion over the distended intestines, is in ascites above the fluid; in ovarian tumor, to the side and behind. When fluid in the cavity coincides with a tumor, the latter may be felt by suddenly displacing the fluid, and coming down on the tumor with the hand. An exploration through the rectum, by the method of Simon, will enable a diagnosis to be made at once; by conjoined manipulation through the vagina, a tumor can usually be easily defined. In pregnancy the tumor develops in the middle line of the abdomen with an inclination to the right; it is firm, inelastic, and non-fluctuating. Changes in the length, density, and size of the neck of the uterus, and in its functions (arrest of menstrual flow), and in the mammæ, with the other evidences of pregnancy, accompany the growth of the uterine tumor. After the fourth month the sounds of the fœtal heart and the placental souffle, together with the *ballottement*, indicate the nature of the case without doubt. The author has known a distended bladder mistaken for ascites. Applying the same method already described for the diagnosis between ovarian tumor and ascites, the difference becomes at once apparent. In all cases of critical examination of the pelvic organs, the catheter is used, or ought to be, to prevent error and to facilitate the exploration. The local and physical signs may be precisely the same in ascites and chronic peritonitis, but the clinical history is so different that a differentiation may be made by reference to the origin, causes, and symptomatology of the two affections. Peritonitis is accompanied by pain and tenderness of the abdomen, by an increased thickness of the walls, by persistent vomiting, and by alternating constipation and diarrhœa; in ascites there is usually no tenderness, the walls of the abdomen become very thin from absorption of fat and atrophy of the muscles, there is no vomiting except such as is due to hepatic disease, and there is persistent constipation. The spleen may be uniformly and extensively enlarged so as to fill the cavity, but it differs from ascites in the following particulars: The enlargement is from the left hypochondrium downward; it is firm, inelastic, and non-fluctuating; the dullness maintains with the tumor a constant position, which does not follow the movements of the patient.

Treatment.—There are, besides artificial means, two outlets to the effusion—by the intestinal canal; by the kidneys.

Dry diet has, from the earliest period, been regarded as a most efficient plan of treatment. As it may be tried without interfering with the remedial management proper, it should be enforced in suitable cases. Dry diet consists in absolute disuse of fluids of every kind, and the use of water-free food. It is extremely irksome, but, if patiently carried out, will contribute materially to relief or cure, as either may be practicable. If this method be unavailable, the opposite plan, or the free use of water and diluents, should be enjoined. The best of all diluents for this purpose is skimmed milk, which should be taken with regularity and in as large quantity as the patient can bear. An intelligent medicinal treatment of ascites must be conducted with reference to its cause. Here only the remedies for the removal of the effusion can be discussed. As the cavity is a closed sac, diuretics are not very efficient. The treatment by hydragogue carthartics is the most generally serviceable, and of the remedies belonging to this group the most useful is the compound jalap powder. Several watery evacuations must be passed daily to make any important impression on the effusion; this result is most easily accomplished by the administration of one or two drachms of the compound jalap powder in the early morning, to avoid interference with the digestion. If the jalap is not efficient, elaterium may be substituted; but in the author's experience the former is to be preferred. Notwithstanding the little utility of diuretics, advantage should be taken of any good arising from them. The resin of copaiba, according to Wilks, is an efficient diuretic in this disease. Cream-of-tartar is also useful. Digitalis, especially in the form of infusion, is the best of the diuretics proper. These remedies may be given jointly. To urge the kidneys to their highest activity, the functions of the skin should not be excited, and the cutaneous capillaries must therefore be kept contracted by lessening the warmth of the covering or clothing. An increased action of the skin is generally more serviceable in ascites than diuretics are, unless an obstructive cardiac or pulmonary disease is the cause of the effusion. Most excellent results are now obtained from the use of jaborandi or pilocarpine in the treatment of ascites. Warm clothing, vapor-baths, and pilocarpine may be used jointly, to maintain constant diaphoresis. Removal of the fluid by tapping is a useful expedient in cases not relieved by the methods advised, but so rapidly does reaccumulation take place that this measure should not be practiced too early. It should not be adopted until the embarrassment of breathing is so great as to prevent sleep. The relief it affords is immense, and is accomplished now so readily that there is a constant temptation to employ the aspirator trocar before the proper time has arrived. The puncture is made in the middle line—the *linea alba*—two or three inches below the umbilicus. It is not necessary to draw off all the fluid, but a sufficient quantity to afford relief. The puncture should be carefully closed. It is sometimes difficult to do this, and the ascitic fluid is permitted to drain away indefinitely; but the practice is bad, for the admission of air to the cavity sets up a septic process, and may excite a fatal peritonitis, as the author has seen.

IDIOPATHIC SUPPURATIVE PERITONITIS is a term applied to a form of peritonitis apparently arising from exposure to cold, and occurring in children. It has the clinical history of peritonitis—sudden onset, fever, small pulse (dicrotic), rapid decline in strength, pain in the abdomen, meteorism, nausea and vomiting, constipation, vesical tenesmus. Pus may be evacuated through the rectum, bladder, vagina, or externally. It is in a high degree probable that the peritonitis is not a primary but a secondary affection, and is due to perforation. The enormous accumulation of gas and its extreme fetidity lend support to this view. Other cases having similar symptoms, and terminating by the discharge of matter, may be examples of the subperitoneal phlegmon.*

DISEASES OF THE PANCREAS.

PRELIMINARY OBSERVATIONS.—So little is definitely known of the diseases of the pancreas that many systematic writers omit the subject entirely. Recently, however, Dr. Fitz, of Harvard Medical School, has demonstrated some new facts of great value, to which reference is made further on. There are, however, some practical points which should receive attention. The pancreas has an office in connection with the digestion of certain kinds of foods. Like the salivary secretion, the pancreatic fluid transforms starch into dextrine and grape-sugar. Although its ferment loses its activity in the presence of an acid, yet the pancreatic juice has the power to complete the digestion of peptones that have escaped final action of the gastric juice.* The emulsionizing, or preparation of fats for absorption, is another function of the pancreatic fluid. It therefore supplements the action of all the digestive juices.

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* See the paper by M. le Dr. Besnier, "Arch. Gén. de Méd.," September, 1878.

Still more important is the action of the pancreatic juice on the bile, whereby certain changes are affected that give distinctive characteristics to the intestinal contents. Appearances that were supposed to be due to the absence of bile or to defects in its composition are now known to be due to certain transformations effected by the pancreatic fluid.

Recent researches by the distinguished American pathologist—Dr. Fitz, of Boston †—have thrown a great light on the morbid anatomy of the pancreas, and have added much to our previously imperfect knowledge of its clinical symptomatology. It may be said, indeed, that all previous studies, fragmentary as they were necessarily, but still constituting the body of our information, have been quite overshadowed by these accurate and painstaking investigations. Besides the new knowledge thus contributed to pathology, it is quite certain that Dr. Fitz has put into our hands new clinical methods for the determination of hitherto unknown morbid states of the organ, and these facts, in turn, give new importance to its physiological relations.

Topography.—The pancreas lies between the xiphoid appendix of the sternum and the umbilicus, extends transversely about ten inches, and is covered in by the lower border (greater curvature) of the stomach and the omentum. The head, so called—at or near the junction of the epigastric with the right hypochondriac and iliac regions—comes into relation to the duodenum, ascending colon, liver, gall-bladder and common duct, and right kidney. Unless in the case of considerable emaciation, or enlargement of the organ, the pancreas can not be felt. When felt it is hard, unyielding to pressure, but a little mobile in position, and on percussion yields a dull, even flat note ; but the presence of much flatus will materially modify this result. The pancreas will have an apparent pulsation, in some conditions, when it comes to lie on the aorta.

HÆMORRHAGE AND INFARCTION OF THE PANCREAS.—We owe our information on this subject to the studies of Dr. Fitz, above mentioned. Now that the condition has been explained, we may well wonder that it has been hitherto a totally unknown subject. When hæmorrhage occurs, much depends on the size of the vessel giving way; but, as a rule, only the smaller arteries are affected. When hæmorrhage occurs in the substance of the pancreas, there is sudden and severe pain of a very depressing kind, for it radiates through the solar plexus, and by a reflex has an inhibiting effect on the heart, which becomes slow and feeble. In fact, we find that there appears the usual symptoms accompanying blows on the epigastrium. The changes that follow the hæmorrhage are first those occurring in blood-clot, and second those signifying an interruption of the functions of the pancreas. When the trypsin of the pancreatic fluid, and probably some other constituents, fail to be produced, the stools change in character, are offensive, party-colored, contain much mucus, and also fats appear in quantity determined by the interference of the disease with the making of chyle. As the nutrition fails, the color fades into an unhealthy pallor mixed with brownish tints and spots here and there, the strength rapidly declines, the heart's action grows more and more feeble, and before long death ensues from exhaustion.

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* Dr. W. Kühne, Virchow's "Archiv," Band xxxix, p. 130.

† Lectures by Dr. Fitz, Pathologist Harvard Medical College, "Medical News" for 1889

A hæmorrhage of sufficient size may kill in a few hours.

In the more chronic cases, there may have been mere extravasation, the symptomatic disturbance is less pronounced at the time, and the various symptoms mentioned above appear in slower manner.

PANCREATITIS.—In the acute form, the changes consist in hyperæmia, increased size and density of the organ, and, it may be, hæmorrhagic extravasation. The inflammation proceeds to suppuration in a portion of the cases, at first in isolated depots, which may subsequently coalesce, forming a large one. Peritonitis may arise when the superficial parts of the organ are occupied by abscesses, and gangrene and sloughing may ensue when there is considerable hæmorrhagic extravasation. Almost nothing is known in regard to the causes of the disease. Men seem to be more frequently affected than women. As pancreatitis seems to have occurred more often several centuries ago, it is highly probable that the excessive use of mercury was an efficient cause. As the functions of the pancreas are merely auxiliary, it is not surprising that but few symptoms are produced when the organ is the seat of an inflammation. Pain, becoming very acute and depressing, is one of the earliest symptoms; it is felt in the epigastrium, and radiates to either shoulder and to the back; there are restlessness, precordial anxiety, faintness, nausea, and vomiting. After much straining, some bilious-looking watery fluid is brought up, but this does not afford relief. There is considerable gaseous distention of the abdomen, and a good deal of gas comes up by eructation. Constipation is also a symptom.*

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* Oppolzer, "Über Krankheiten des Pancreas," "Wiener med. Wochen.," 1867, No. 1.

From the beginning there is fever; the pulse, at first full and tense, soon becomes small, feeble, and irregular. The symptoms of depression make rapid progress, and in a few days (four to six) the patient is in a condition of collapse, with shrunken features, cold surface, cold extremities, and failing heart. The marked anxiety and depression from the first and the weak and irregular action of the heart indicate an implication of the solar plexus; for similar symptoms are produced artificially (crushing-blow experiment). It will be difficult to distinguish this affection from hepatic colic, or gastralgia, except by the fever, the rapid and irregular action of the heart, and the early collapse, which are wanting in these two disorders, which also terminate in a few hours—one with jaundice and returning health, the other with complete relief and immediate resumption of the functions. The termination, after a very rapid course, is usually in death; but there may be a gradual decline into a chronic state, ending in abscess or slow induration. Acute pancreatitis may be secondary to other affections—there may occur in it, during the course of acute infectious diseases, the changes included in the term parenchymatous degeneration.

SUBACUTE PANCREATITIS, according to the author's observations, confirmed by Dr. Earle,* is a by no means infrequent affection. A typical case, ending in apparent recovery, and therefore not confirmed by *post-mortem* examination, furnishes the principal data on which the following description is based. Since the symptomatology agrees in the main with the clinical features of the acute and chronic, or sclerotic form, it may be assumed with a high degree of probability that the diagnosis was correct. Various cases have, from time to time, come under my charge, but that above referred to seems to be the most complete. I supplement my own with the careful observations of Dr. Earle, and I avail myself, also, of the classical account of Oppolzer.

The symptoms are local and systemic. The former consist in pain and tenderness along the pancreas, and an amount of swelling easily recognized in thin subjects. The pain is rather a sense of distress combined with heat, which radiates in all directions, but especially upward into the chest, and backward under the scapulæ. This uneasiness or pain is accompanied by a sense of depression about the precordial region, and an actual slowing and weakening of the heart's action. At certain intervals, by no means regular in recurrence, paroxysms of a less or more severe character occur, during which the distress in the region of the pancreas—the soreness and uneasiness—are considerably increased above the ordinary level of suffering, the heart's action becomes very feeble, the surface of the body grows cold and is covered by a clammy sweat, and hebetude of mind, or melancholic depression, comes on with the other symptoms.

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* "Cirrhosis of the Pancreas," "New York Medical Record," November 8, 1884.

The digestion is impaired ; the food lies heavy, and constipation or diarrhœa occurs, but the former is more common, and they may alternate. The appetite is poor and capricious ; nausea is not usual, vomiting is rare, but the sensation of epigastric distress is accompanied by squeamishness. The stools are not healthy—have a pronounced and rather offensive odor, are dark in color usually, scybala are mixed with liquid or semi-fluid material, and they may be sour-smelling, acrid (presence of butyric acid), yeasty, and mixed with merely decomposing matters. An excess of fat may or may not be evident; in my experience usually not—in this respect differing from the stools in cancer of the organ.

The nutrition of the body is lowered in consequence of the insufficient appetite, the feeble circulation, and the impaired intestinal digestion. The face has an anxious expression, the skin a sallow tint, often pigmented in spots, and the mucous membrane of the lips and cheeks is pallid, and the tongue is somewhat pointed, irregularly glazed and dry, and but little coated, as a rule. Some salivation is a common symptom ; occasionally it is profuse.

The duration of this affection is rather indefinite. After several weeks or months, an improvement in the general state takes place, the swelling and tenderness subside, the intestinal discharges assume a natural aspect, and ultimately health is restored.

The termination of this disease is probably more often in interstitial pancreatitis, or sclerosis of the pancreas. When this occurs, the symptoms are similar in character to those of the subacute form, but are less sharply defined.

The *chronic interstitial pancreatitis*, affecting parts of the gland, is the form which the chronic inflammation most usually takes. The connective tissue undergoes hyperplasia, and the proper gland-structure wastes. When the whole organ is involved, there may be an entire disappearance of the proper gland-structure, or a part of it may be converted into a connective-tissue bundle. As in cirrhosis of the kidney, cysts are produced by obstruction of the ducts. Calculi form in the ducts, and the duct of Wirsung may be entirely occluded by a calculus, inducing dilatation of the ducts and atrophy of the gland-substance. Abscesses may also result from the pressure and inflammation caused by calculi. Chronic parenchymatous pancreatitis is a less usual form of chronic inflammation. It is probably more frequently secondary than primary—i. e., due to the extension of suppurative inflammation from neighboring parts.

The treatment must be entirely symptomatic. Pain must be relieved by morphine hypodermatically, the stomach symptoms by carbolic acid, bismuth, pepsin, ingluvin, hydrocyanic acid, etc., and the chronic interstitial change is best treated by minute doses of corrosive sublimate, iodide of potassium, and similar remedies.

CANCER OF THE PANCREAS.—Much more is known in regard to this than to any other affection of the pancreas. The ordinary form of cancer affecting this organ is scirrhus, and scirrhus characterized by a denser stroma. Medullary and colloid have also appeared in the pancreas, but very rarely. Scirrhus of the pancreas is more frequently secondary than primary, and even as a secondary disease it is very rare, occurring in cancer cases in the proportion of about six per cent. only. It develops most frequently in the head of the pancreas and occurs there as a secondary disease, and extends thence over the body of the organ. It is more frequently confined to the head than to other parts of the organ ; in 200 cases there were 33 in which the disease was confined to the head, and in 88 the whole organ was affected.* A tumor of the pancreas of considerable size must impinge on neighboring organs ; it may compress the ascending vena cava, causing œdema of the lower extremities ; the ductus communis choledochus, causing jaundice ; the pancreatic duct, causing dilatation and the formation of concretions ; the ureter, causing hydronephrosis, and the duodenum, causing stenosis and dilatation of the bowel above and subsequently of the stomach. It is usual for cancer of the pancreas to extend to and implicate other organs, which may be bound down into a uniform mass, in which the point of initial deposition may not be distinguishable. The duodenum, the stomach, the gall-bladder, the kidney, the liver, mesenteric glands, and peritoneum may all be included in a mass of which the beginning was in the head of the pancreas. Ulcerations into neighboring organs may also take place—as into the stomach, duodenum, vena cava, portal vein, splenic artery, etc.

Cancer of the pancreas is more frequent in males than in females ; in Dr. Da Costa's† cases there were 24 males and 13 females : nearly twice as frequent, which is the proportion noted by other observers. As is the rule with scirrhus in all situations, the morbid growth makes its appearance from forty to sixty years of age. Pain is an early symptom, and, as it appears without cause, is persistent and rather increases than diminishes, and as progressive emaciation and feebleness accompany it, especially if the age of the subject be suitable, it is extremely suggestive of malignant disease. The pain is situated in the epigastric region and radiates through the numerous ramifications of the solar plexus, into the back, through the abdomen ; it is pretty constant, with paroxysms of great severity in which the suffering is agonizing ; it is increased by the erect posture, and is relieved by bending the body forward. The presence of a tumor has a high degree of importance, but it is not always found, and when discovered may be misleading. A tumor is discovered in not more than one third of the cases, owing to the depth at which the pancreas lies. The head of the pancreas has been often mistaken for scirrhus. If enlarged lymphatics be felt, and especially if the cervical lymphatics are enlarged, support will be given to the supposition that an existing tumor is malignant. In a small proportion of cases, an excess of fat in the stools is a symptom which throws light on the case. The appearance of jaundice, the passage of blood by stool, œdema of the lower extremities, and disorders of digestion, are coincident with the extension of the new growth to neighboring organs, and rather confuse than clear up the diagnosis. In Da Costa's 37 cases, jaundice was present in 24, dyspepsia in 25, dropsy (anasarca or ascites) in 15. With the development of these symptoms there is a corresponding increase in the gravity of the constitutional state. The general condition and the cachexia, such as have been described as belonging to cancer of the stomach, are present in these cases. The duration varies somewhat. The most severe terminate in a few months, and but rarely is any case protracted beyond a year. The rate of progress is influenced by the complications—by the pressure on neighboring organs and interference with their functions. Sudden death may be due to erosion of a large vessel.

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* Ancelet, "Études sur les Maladies du Pancreas," Paris, 1866, p. 34.

† "N. A. Med. Chirurg. Review," September, 1858, p. 883.

CYSTS OF THE PANCREAS.—Chronic interstitial pancreatitis is the chief factor in their causation, as in the production of the corresponding cysts of the kidneys. Ducts being obstructed by the growth of the connective tissue (hyperplasia), the contents of the acini—the secretions—accumulate, the walls yield to the increasing pressure, and thus a cyst is formed. Hæmorrhage into such cysts, purulent transformation, and albuminoid degeneration, effect important changes in the contents of these cysts. Obstruction of the duct of Wirsung by a calculus, by neoplasms, by cancer of the duodenum and tumors, will cause a cystic degeneration of the whole gland.

CALCULI OF THE PANCREAS.—These are concretions, consisting of carbonate and phosphate of lime, which have crystallized about a bit of inspissated mucus or other organic matter. To produce them there must be a catarrhal state of the mucous lining of the ducts, a change in the secretion toward an excess of its earthy constituents, or an obstruction leading to retention of the secretion. The pancreas is also liable to amyloid and fatty degeneration, and is sometimes the seat of secondary tubercular deposits.

DISEASES OF THE LIVER.

TOPOGRAPHY OF THE LIVER.

THE liver, with its annexed apparatus, occupies the right hypochondrium, and extends in part across the epigastrium (Fig. 13). On percussion the area of the hepatic dullness is determined by the position of neighboring organs, as well as by those variations in the size of the liver due to disease and to its own normal elasticity. Under variations of the blood-pressure—as, for example, in mitral obstruction or regurgitation, and in portal stenosis—the size of the liver increases or lessens. Distention of the intestines with air, and emphysema of the lungs by increasing sonority at the borders of the liver, narrow the boundary of the dullness. When the patient is recumbent, the liver gravitates upward and backward; when the patient is upright, it glides downward and forward—hence the area of dullness shifts correspondingly. Besides the variations in the area of dullness due to these several factors, the vertical line of dullness differs at different points: in the axillary line it is four to five inches, and in the mammary line three to four inches. There is a difference between the deep and the superficial dullness. On strong percussion the deeper parts are thrown into vibration, and hence the wider the area of dullness; whereas on slight percussion the shelving margin of the lung vibrates alone, thus lessening the dullness.

In health the inferior border of the liver extends to the margin of the ribs. If on palpation it can be felt beyond, there is probably enlargement of the organ, unless effusion into the thorax or a tumor, etc., depress it downward. In the axillary line the dullness extends to the upper border of the eleventh rib (Fig. 13, XI). Besides the various conditions external to the organ which may affect its position, it should not be forgotten that—as has been stated—the liver has a considerable

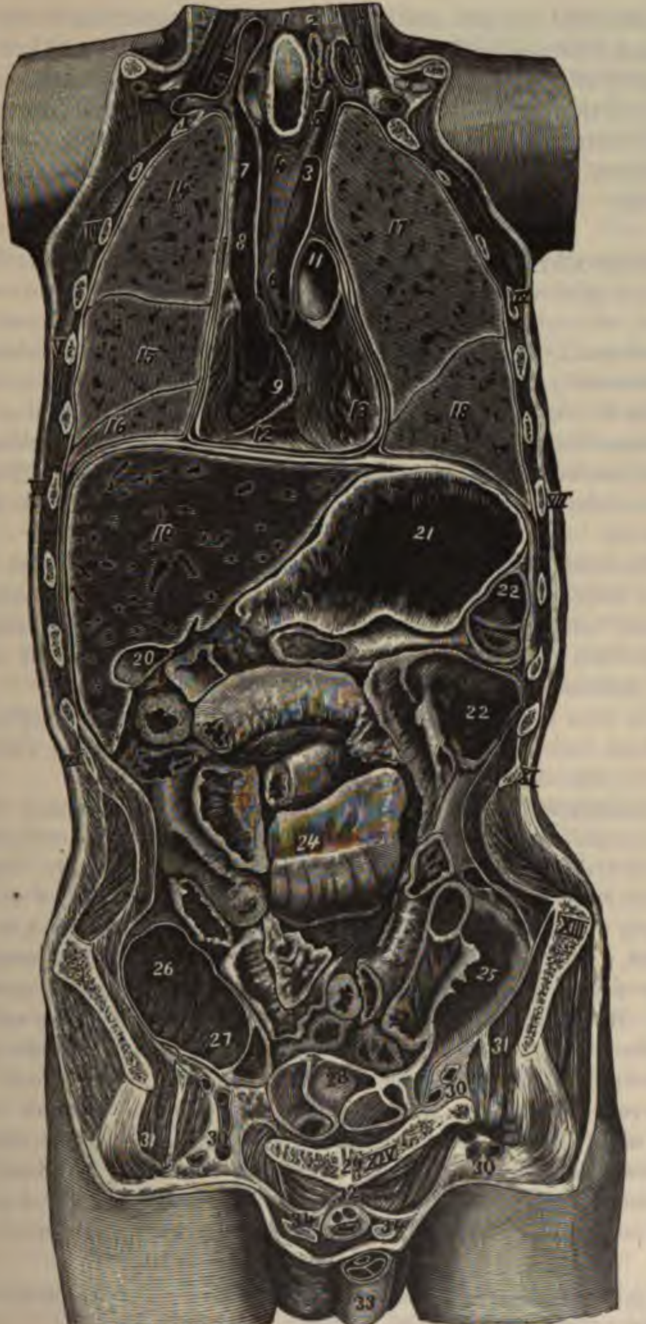

Fig. 18.—Horizontal Section of Thorax, Abdomen, and Pelvis, Frozen.—1, Section of Trachea 11, Left Auricle; 13, Left Ventricle; 9, Right Ventricle; 19, Liver; 20, Gall-bladder; 21, Stomach; 26, Cæcum; 27, Entrance to Appendix; 25, Descending Colon. After Rüdinger.

elasticity, and expands and contracts with the variations in the amount of blood contained in it. Mere change of position should not be mistaken for alteration in size. It is a good plan, in exploring the topography of the liver, to fix first the upper border of the organ, which should be marked with ink, and then ascertain the inferior margin by percussion from below upward until the line of hepatic dullness is reached.

COMPOSITION OF AND TESTS FOR BILE.—The bile is a fluid having a golden, reddish-brown, or greenish color, and when free from mucus, without viscidity. In reaction it is faintly alkaline or neutral—most usually the latter—and it has a specific gravity of about 1010, when recently secreted; but if it is retained for some time in the gall-bladder it becomes darker in color, viscid from the presence of mucus, and more distinctly alkaline. The quantity of solids is variously estimated, according to the time it has remained in the gall-bladder—from two to three per cent. when fresh, and rising to ten to fifteen per cent. when old.

The solids of bile consist of a coloring-matter or pigment—*bilirubin* or *biliverdin;* of peculiar acids, combined with soda—glycocholate and taurocholate of soda; of fatty matters—cholesterin, soaps, and lecithin; and of salts—chloride of sodium, phosphate of soda and lime, carbonate of sodium, etc.

The tests for bile when mixed with other animal secretions have not been satisfactory hitherto, nor are they now unless, indeed, Dr. Oliver's peptone test prove sufficient.

Gmelin's test for bile-pigment consists in the reaction with nitric acid containing some nitrous, which is the case with the ordinary commercial article. There are several modes of applying it. The most distinctive is to place on a porcelain plate a thin stratum of urine, and bring in contact with it some nitric acid. At the point and instant of contact, a play of colors due to the oxidation of the pigment takes place—first, a green, then blue, violet, and red zones successively appear. Rosenbach suggests the plan of filtering the urine, and touching the pigment adherent to the paper with a drop of nitric acid—a green circle forming at the point of contact. Hydrochloric acid is preferred to nitric by Harley. This, brought into contact with the urine on a white plate, gives to the pigment an olive-green tint.

The most promising test for bile when contained in mixed animal secretions is that of Oliver.* It is based on the fact that an acidulated peptone is precipitated from its solution on the addition of bile.

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* London "Lancet," April and May, 1885, "A Contribution to the Clinical Study of the Liver viewed through the Urine," by George Oliver, M. D., London.

The test solution, as recommended by Oliver, has the composition as follows: "Pulverized peptone (Savory and Moore), thirty grains; salicylic acid, four grains; acetic acid (B. P.), thirty minims; distilled water, eight ounces. Perfect transparency is obtained after repeated filtration." The reaction is described by Oliver as follows: "When twenty minims of urine, containing bile salts in pathological quantity, are run into sixty minims of the test solution, an opalescence appears proportionate to the amount of bile derivative." This test is not only exceedingly delicate, but there do not appear to be any sources of fallacy in respect, at least, to the ordinary constituents of the urine.

Pettenkofer's test can not be used with any degree of certainty for bile in complex animal solutions, such as urine, but for bile alone it satisfies all the requirements. It is applied as follows: In the solution of bile some strong sulphuric acid is put, and when the bile acids are precipitated more sulphuric acid is added, until they are dissolved. A little sirup is now dropped into the liquid, and the whole is gently warmed, when a fine purple color like that of a solution of permanganate of potassium is developed. Strassburg's modification of this test is sometimes convenient. To the mixture suspected to contain bile—urine, for example—a little cane-sugar is added, and a strip of filtering-paper is dipped into it and then dried. A drop of strong sulphuric acid is now let fall on the paper, when, in less than a minute, a ring of violet appears around the drop.

SPECIAL DISEASES: JAUNDICE, OR ICTERUS.

Definition.—The term *jaundice*, derived from the French word *jaune*, signifying yellow, means a yellowish discoloration of the skin due to the deposition of bile-pigment. The Greek word *icterus*, sometimes used to designate this state, has the same meaning. Jaundice is a symptom, common to many affections of the liver. To give it separate consideration, as if it were a disease, is not, therefore, a logical position, but it is the usual practice of systematic writers, and the author is, therefore, constrained to follow it, that his work may not appear deficient.

Etiology.—There are three conditions under which jaundice may be produced. Hæmatogenous is that due to the disorganization of the blood and the separation of hæmatoidin. At one time bilirubin—a bile-pigment—and hæmatoidin—blood-pigment—were supposed by the physiological chemists to be identical. It would hence follow that, were any substance to enter the blood capable of setting free the coloring matter, the appearance of jaundice would result. The bile acids were supposed to effect this change, setting free hæmatoidin, and thus causing hæmatogenous jaundice. But the identity of the two pigments is no longer admitted, and the production of artificial jaundice has not been accomplished by injecting bile acids into the blood. The theory of hæmatogenous jaundice is, therefore, no longer tenable, with the data at present in our possession.

The existence of a jaundice by suppression was entertained for several centuries, and has been supported by eminent authorities of the present generation—by Watson,[*] Bamberger,[†] Budd,[‡] Trousseau,[‖] Moxon,[§] and others—but is now generally abandoned except by Harley, who in the last edition of his work [1885] reaffirms and vigorously maintains his original position. To establish this theory, it is necessary to show that the bile exists preformed in the blood, and that the liver merely acts as a strainer, to separate it. Notwithstanding the detection of the bile acids is comparatively easy, neither acids nor pigments can be found in the blood—not even in the blood of the portal vein. Hence it follows that jaundice by suppression does not exist.

The third theory, which is universally admitted to be true, if it may be by some regarded as limited in application, ascribes jaundice to the reabsorption into the blood of bile already formed by the liver. There are two conditions under which this is known to occur, and a third, which is supposititious. The usual condition consists in an obstruction of the ducts. Heidenhain has proved experimentally that a very slight obstacle suffices. It has been observed clinically that jaundice occurred when there was such a slight catarrhal swelling of the intestinal extremity of the common duct as left no trace after death, except the staining which marked the limit of the bile-flow.

Again, bile will flow into the vessels instead of into the ducts, when the pressure in the former is lower than it is in the latter. This, Frerichs maintains, is the chief cause of jaundice, and Heidenhain has also proved it by direct experiment. The occurrence of jaundice by mental emotion can only be rationally explained by this theory. It has been maintained that jaundice may be produced by the absorption of bile that the intestines fail to dispose of. A species of circulation of the bile is, by Schiff, maintained to exist. That which is cast into the duodenum from the common duct is taken up by the radicles of the portal vein, undergoing changes not well understood, but probably due to an action of the pancreatic juice, as is the present notion, and is again excreted by the liver. A failure to destroy this portion of the bile, or the presence of, or an excess of, may result in the absorption of sufficient to produce jaundice. There is little probability in this theory.

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[*] "Lectures on the Practice of Medicine," Lecture lxxv.

[†] "Krankheiten des chylopoietischen Systems," p. 517.

[‡] "On Diseases of the Liver," p. 373.

[‖] "Clinique Médicale," tome iii, p. 274.

[§] "Transactions of the Pathological Society."

Pathological Anatomy.—The liver being most concerned should be carefully examined. Obstruction of the ducts by hyperæmia and catarrhal swelling may disappear after death. In that case, the presence or absence of bile-staining will indicate the seat of the obstruction. Very often the *ante-mortem* state is shown to have been an obstruction at the duodenal orifice of the common duct, by the absence of bile-staining here and in the neighboring part of the intestine, while above in the ducts the staining is well marked. A plug of mucus in the common duct, not stained with bile, will show that no bile has passed since the formation of the plug. Masses of epithelium and mucus may form obstructions high up in the smaller ducts. The hepatic or common duct may be obstructed by a calculus. Tumors, enlarged lymphatics, an aneurism, cancer of the pancreas, etc., may occlude by exterior pressure. When the obstruction, of what nature soever it may be, is persistent, important changes occur in the liver. The ducts and gall-bladder become enormously dilated—in a uniform or sacculated manner. The walls of the duct thicken, due to a hyperplasia of the connective-tissue elements, and the follicles dilate. The coloring matter of the bile present in the ducts is finally absorbed, and only a colorless fluid remains. The substance of the liver changes, has a mottled appearance due to the tinting of the center of the lobule with a deep yellow, while the peripheral portion has a lighter yellow shade—changes due to the deposit of pigment. The bile when the ducts are obstructed passes out of the liver by the lymphatics into the thoracic duct, and thence into the blood (Fleischl).* The obstruction continuing, the depth of color in the lobules increases, changing to a dark green finally; the substance of the liver becomes firmer and granular, enlarges a little, and then begins to shrink. If the jaundice has persisted for a long time, a considerable overgrowth of connective tissue takes place, the hepatic cells shrink, become angular, and their contents have a shining, rather vitreous appearance, and in some cases are destroyed.

The kidneys also undergo important changes in jaundice, as Frerichs was the first to show. When the jaundice has been of long standing, the kidneys have a deep-olive tint, the tubules are stained green or brown, and the epithelium is fatty, colored greenish, brownish, or black, and is largely detached. In extreme cases, the deposits of pigment fill the tubules with a black mass.

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* Legg, "On the Bile, Jaundice, and Bilious Diseases," New York, D. Appleton & Co., 1880, p. 351.

Symptoms.—Jaundice first appears in the conjunctiva, and immediately the bile-pigments may be detected in the urine by Gmelin's test. Then the skin of the face appears sallow or fawn-color, quickly changing into yellow, and thence the yellowness extends to the chest and arms, to the abdomen and lower limbs, and finally the whole body is uniformly yellow. In slight cases, the face and thorax only may be tinged. The mucous membrane of the mouth is stained ; but it does not show in the lips, because of their redness, and is visible in the roof of the mouth and the soft palate. Very rarely does the saliva exhibit a yellow tinge, and present the reaction for bile-pigment ; but the milk of the nursing mother, jaundiced, contains bile, and jaundice is said to be communicated to infants in this way. In a very short time, within forty-eight hours probably, bile-pigments appear in the blood ; but doubts may be expressed in regard to the presence of bile acids, although they have been discovered in the blood of dogs whose common duct had been tied. Becquerel and Rodier, and afterward Frerichs, found that there was a large increase of fatty matters and cholesterin. The blood is also changed, as respects the red globules, which are diminished in number. The urine early undergoes a change, and becomes intensely colored with bile-pigment, which it imparts to linen and white paper dipped into it. The color varies from a simple increase in the depth of the normal color up to the darkness of black coffee. The usual tint is that of dark sherry or brandy. The urine is usually turbid on cooling, from the presence of a great quantity of urates ; is acid in reaction, and the specific gravity is well up toward normal. Albumen, at least in traces, is rarely absent from the urine in jaundice. The best test for the presence of bile in the urine is the nitric-acid or Gmelin's test. It is best performed by pouring into a test-tube about an inch of nitric acid ; then let drop from a pipette some suspected urine on the side of the tube. As the urine comes in contact with the acid, if free from bile, it forms a red line on the margin of the acid ; but if it contain bile there will be an alternation of colors—green, blue, violet, and red—the green being uppermost. Although the bile acids have not been found in the urine, nitrogenous derivates from them were discovered by Hoppe-Seyler, Kühne, and Bischoff. Recently minute traces of the bile acids have been detected in the urine in health. It is supposed that absorption of these takes place in the intestine. If jaundice has long continued, the bile acids cease to be produced, and hence are not to be found in the urine. The amount of urea present in jaundice may be normal, or above or below normal, so that at present no conclusions can be drawn from this fact. Casts are sometimes present in the urine, especially hyaline, sometimes epithelial.

The stools in jaundice are grayish or slate-colored, sometimes quite white. In other cases, portions of the stools are stained with bile, some parts not containing any, giving them a parti-colored appearance.

They are often very fetid, from the decomposition of some articles of food, and the wind passed is equally foul. Again, they may be entirely without odor. As a rule, the bowels are rather constipated, although diarrhœa may occur.

Jaundice is usually unaccompanied by pain. It is true, those cases due to the passage of gall-stones are preceded by the most severe suffering; but the jaundice itself is comparatively painless. Headache is a common symptom; but it is rarely considerable—often only a tensive feeling in the frontal region. Drowsiness, hebetude of mind, and despondency are commonly experienced. The appetite is generally poor, but is sometimes inordinate or canine. Nausea, a persistent and harassing hiccough, and a heavily furred tongue, are present in some cases; in others, the digestion is good and the tongue clean. Languor, muscular soreness, inability for any considerable exertion, and decline in the bodily vigor generally, are experienced in most cases. As that important material, glycogen, ceases to be formed when the bile-ducts are obstructed, an adequate explanation is thus afforded of the decline in strength. Although various disturbing causes may alter the conditions, in jaundice the temperature is barely normal, or is below normal. It has been shown experimentally that the injection of the bile acids into the blood lowers the temperature. In those diseases—as pneumonia, for example—in which high temperature is the rule, the fever-heat does not attain the usual altitude when jaundice occurs. It sometimes happens that the temperature rises quite high—104°, 105°, or 106°—at or near the termination of chronic jaundice; but this is indicative of a fatal termination, and is due to or accompanied by a considerable disturbance of the nervous system.

In many cases of jaundice the pulse is slow. Frerichs has met with a case in which it was twenty-one to the minute, but it rarely descends below fifty. Exercise and the occurrence of inflammatory action increase the pulse-rate when it is abnormally slow. The cause of the slow beat has been variously interpreted: it has been ascribed to increase of inhibition; to the action of the bile acids on the cardiac muscle and on the cardiac ganglia. A murmur has also been discovered in the mitral area in the cases of slow beat of the heart, due, it is supposed, to diminished power in the papillary muscle, and whence imperfect action of the mitral-valve segment.

More or less itching of the skin, sometimes an intolerable itching, is observed in many cases—probably in three fourths of the cases. It is sometimes preceded by nettle-rash; and the itching may precede the jaundice by several days, even weeks. Sometimes the itching ceases when the jaundice appears; but it comes on in most cases with the jaundice, and gradually declines. The itching is usually worse at night, and is most annoying where the clothes have pressed. The taste in jaundice is often bitter, and sapid articles are not rightly appreciated. Rarely is the vision yellow, all objects being seen through a yellow medium.

In the more ancient cases of jaundice, yellow, leathery patches, round or oval in shape, appear on the skin, especially on the eyelids and hands: they are entitled *xanthelasma*, and two forms are recognized—the flat and the tuberous. The change consists in a fatty infiltration of the affected area. It is not necessary to the appearance of this affection, that the jaundice be intense, but it must be long continued. It very rarely arises earlier than four months, and usually not earlier than a year, after the appearance of the jaundice. Why it should develop in some cases of jaundice and not in others is unknown. The conditions exciting it are equally obscure. It may slowly disappear without leaving any changes, but remedies do not affect it.

Course, Duration, and Termination.—The behavior of jaundice is so largely dependent on its cause, that no course can be laid down applicable to all cases. The jaundice of acute yellow atrophy and of phosphorus-poisoning is of short duration and fatal, while that due to a permanent obstruction is most protracted. The jaundice arising from catarrh of the ducts depends on local conditions, which vary in extent and importance. The reader will find under the several maladies of the liver the manner in which the course, duration, and termination are thus influenced.

Diagnosis.—A simple inspection suffices to make the diagnosis of jaundice, but to determine its cause is most difficult. If the jaundice comes on in the course of a gastro-duodenal catarrh, and there is no apparent change in the liver, nor disease of other organs, it is probably a case of simple catarrhal jaundice. If it occur in the course of a malarial fever, it is probably of this form. If the jaundice is preceded by sudden violent pain in the right hypochondrium, it is due to the passage of gall-stones. If the jaundice be intermittent, but irregularly so, and if the attacks occur frequently, the jaundice from one attack not disappearing before another comes on, the cause is probably gall-stones. If the attacks are regularly intermittent, and accompanied by fever of high grade, preceded by a chill and succeeded by a sweat, the cause is malarial. If the jaundice be persistent—lasting many months—and intense, and follow an attack of pain which has not been repeated since, the liver remaining unchanged in size, it is probably due to a permanent occlusion by a gall-stone. An intense, persistent jaundice, with uniform and painless enlargement of the liver, without other indication of disease, may signify obstruction by hydatids. A faint jaundice, a mere fawn-color, lasting many months unchanged, with evidence of contraction after a period of enlargement of the organ, is produced by sclerosis or cirrhosis, or nutmeg-liver, and this diagnosis becomes more certain if there be present ascites and enlargement of the superficial veins of the abdomen. Jaundice, with persistent pain or soreness in the right hypochondrium and considerable enlargement of the liver, indicates cancer.

Treatment.—The several conditions of which jaundice may be a symptom require treatment according to their nature, and the reader is referred to the special articles for the necessary guidance. Irrespective of the cause the treatment of jaundice is concerned with the removal of this symptom. As most cases are due to some kind of obstruction, it is improper to stimulate the organ when it is unable to discharge into the intestine that which it has already produced. Laxatives and diuretics are the obvious remedies to secure the elimination of the biliary matters circulating in the general system. Grain-doses (one grain) of calomel, rubbed up in sugar of milk, and given at night, is the best laxative in these cases, but this remedy must not be given frequently, lest ptyalism result. Calomel allays the irritability of the gastro-duodenal mucous membrane, lessens the activity of the liver, which is shown by the diminution in the amount of bile excreted, and stimulates to increased effort the glands of the lower ileum and cæcum. This combination of actions renders calomel peculiarly serviceable in the obstructive forms of jaundice. Saline laxatives, which have a diuretic action, are very useful: they promote elimination by the two most important channels. In two instances of persistent jaundice from catarrhal obstruction, I have seen very sudden relief afforded by the compound jalap powder.

Some of the newer antiseptics have proved useful in the treatment of jaundice. Of these, the benzoates and salicylates of sodium and the salicylate of cinchonidine have exerted the most distinctly curative action. Salol, which is a salicylate of phenol, has been highly commended recently for its therapeutical utility in affections of the intestinal mucous membrane characterized by fermentation of the contents of the canal in process of digestion.

In malarial jaundice there can be no question regarding the utility of quinine. The swollen liver, and, also, the swollen spleen, yield rapidly to the action of sufficient doses—from 10 to 30 grains each day.

In gouty jaundice the salts of manganese and permanganate of potassium, sodium phosphate, and salicylate of cinchonidine are useful in a high degree.

CONGESTION OF THE LIVER.

Definition.—By congestion of the liver is meant an increase in the amount of blood in the organ. Owing to the mechanical arrangement of its vessels, the circulation in the liver is influenced by the condition of the heart and lungs, by the state of digestion, and by the action of the diaphragm and abdominal muscles. It is peculiarly liable to suffer from changes in its blood-supply. It may be active (malaria, excesses in eating), or passive (mechanical stasis from obstruction at the heart or lungs).

Causes.—The increased fullness of the portal vein and hepatic artery during the process of digestion is a physiological state, which becomes pathological when excesses in eating and drinking are habitually committed. The admission of irritating substances to the blood, as alcohol, highly stimulating condiments, the salts of lead, phosphorus, etc., increases the tendency to congestion. In malarious regions, congestion of the liver is produced and maintained by the absorption of malaria, especially when in sufficient quantity to cause febrile attacks. Without the objective evidence of malarial infection afforded by fever, the spleen may greatly enlarge (ague-cake), and the liver be kept abnormally full of blood.

Obstruction and regurgitation of the mitral orifice and of the right cavities induce abnormal fullness of the venous system and ischæmia of the arteries. After the lungs, the liver is the first organ to suffer the passive congestion thus caused. The same result is produced when an obstructive disease of the lungs maintains congestion on the venous, and ischæmia on the arterial side of the systemic circulation.

A state of the nervous system may affect the circulation in the liver to a great extent: injury of the semi-lunar ganglion causes immense congestion (Frerichs). Section of splanchnic nerves and the action of curare and some other poisons have the same effect. A fit of anger has brought on an attack of jaundice. Indeed, the facts prove that the nervous system, probably through the vaso-motor nerves, exercises an immediate influence over the circulation of the liver, the mechanism consisting in an increased or diminished blood-supply, by paresis or spasm—by the action of the dilator or constricting fibers of this system. Also, the liver possesses considerable elasticity, and enlarges and contracts with the increase and the diminution of the vascular pressure (Brunton).*

Congestion may also occur in consequence of sudden arrest of an habitual discharge, and has followed a successful operation for hæmorrhoids.†

Pathological Anatomy.—When the congestion is the result of mechanical obstruction at the heart or lungs, the changes which are entitled "the nutmeg-liver" are seen on section of the organ. At the center of each lobule the dilated radicle of the hepatic vein, enlarged and congested, may be discerned, while the neighboring parts of the lobule are pale, and the radicles of the portal are by comparison less full of blood, and really contain less because of the increased pressure from dilatation of the central vein. On section, a greater quantity of venous blood flows out than is normal, and the whole organ is darker and larger. The hepatic cells are either normal or present in places some cloudiness from albuminous infiltration, commencing fatty degeneration, and some brown-pigment deposition (Förster). The compression exercised upon the hepatic ducts interferes with the discharge of bile; and staining of the lobules about the central vein is a result, causing that appearance known as "hepatic icterus." The consistence of the liver is augmented by the congestion if it continue for a lengthened period. The bile is not changed in its composition (Frerichs). A catarrhal state of the ducts is set up as a consequence of the congestion, and in due course hyperæmia of the portal radicles of the gastro-intestinal canal takes place, and a catarrh of the mucous membrane results.

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* "Lettsomian Lectures" for 1885.

† Murchison, "Diseases of the Liver," 1877, p. 134.

Long-continued hyperæmia of the liver establishes a slow atrophic degeneration of the organ, consisting in wasting and disappearance of those cells lying in contact with the dilated central vein, their places being supplied by connective tissue having a granular appearance. The disappearance of these cells and the contraction of the newly formed connective tissue cause a diminution in the size of the liver, and an increase of its density, so that this state is often confounded with cirrhosis; but the substance of the organ has not the density, nor are there present the prominences which give the nodular aspect to the latter.

Symptoms.—Acute congestion of the liver usually begins with a general *malaise;* aching in the limbs and back; some slight rise of temperature toward evening; headache; a coated, yellowish tongue; loss of appetite, even repugnance to eating; nausea. More or less uneasiness, usually a feeling of weight and of tension, and tenderness, are experienced over the hypochondrium; lying on the left side causes a very unpleasant sensation of weight and dragging; buttoning of the clothing can not be borne; and the easiest position is recumbent, with the decubitus toward the right lateral plane, so that the congested organ can be well supported against the ribs. On the other hand, many patients seek a different position and can not bear any pressure against the hypochondrium. On percussion, the area of hepatic dullness is enlarged in all directions. In the normal state the upper border of the liver is parallel with the lower border of the sixth rib on the mammillary line—in ordinary quiet breathing; on full expiration the liver rises on a line parallel to the fifth rib, and on full inspiration it falls to the seventh. The lower border of the liver in health corresponds to the inferior margin of the ribs, or extends a finger's breadth below. If the liver is enlarged by hyperæmia, the hepatic dullness will extend across the epigastrium to the left hypochondrium. It is highly important to note that the area of dullness does not represent the actual size of the organ, for the thin margins do not return a dull sound on percussion. Especially will misconception occur on this point when the ascending colon is distended with gas. Again, the area of hepatic dullness may be greatly enlarged downward by alterations in the form and shape of the liver, when congenital, produced by tight lacing, etc., or displaced downward by effusion in the thorax, tumors, etc. Although percussion affords the most certain physical evidence of enlargement of the liver, inspection may afford some assistance in making a diagnosis, as by the eye an enlargement of the hepatic space may be discerned. By palpation, the liver may be felt projecting below the ribs, and its smoothness or nodulation, its density and resistance, may be readily determined. By mensuration, the diameter of the two sides may be compared, when it will be found, if the congestion is considerable, and the atrophic change has not occurred, that the right is enlarged. A very characteristic symptom in these cases is a light grade of jaundice. If there be no recognizable tinting of the skin, the sclerotic will be distinctly yellow, and the complexion will have the so-called "muddy" aspect. The integument in the cardiac liver is somewhat earthy, faintly yellow, or fawn-color, as in various cachexiæ. In the acute congestion due to temperature changes, to malarial infection, to excesses in eating and drinking, etc., there is usually some gastro-duodenal catarrh, and catarrh of the bile-ducts, and consequently an obstacle to the outflow of bile, with more or less intense icterus. The urine in every case contains some pigment, and varies in tint from pale sherry to a port-wine color, and casts an abundant deposit of urates with much pigment matter. In the more severe cases there is considerable gastric disturbance, and vomiting of bile, and large, so-called bilious discharges take place by the bowels. The stools, after the ordinary fecal evacuations, consist of a greenish-yellow or brownish matter, semi-fluid or thinner greenish or yellowish liquid having the appearance and consistence of stored-up bile. Sometimes a large quantity of such material is discharged, giving great relief, the pain, soreness, and heaviness in the side and the headache and feverishness disappearing. Such acute cases are due to climatic, malarial, or dietetic causes. In the cases of congestion due to cardiac diseases or pulmonary obstruction, the symptoms of hepatic congestion come on slowly; there occur a gradual tension and weight in the right hypochondrium, a slow increase in the size of the liver, an enlargement of the area of hepatic dullness, and, usually, a very slight appearance of icterus, combined with more or less cyanosis, producing a violet-yellow or greenish coloration. Often, in protracted examples of this form of congestion, there exists extensive gastro-intestinal catarrh, with disturbed digestion, nausea, vomiting, diarrhœa, etc. In those cases of congestion of the liver due to psychical impressions, jaundice is the main symptom; there exists really a congestion in biliary production, with more or less hyperæmia, but there is no marked enlargement, tenderness, or heaviness in the hepatic area, and the patients experience the sensations belonging to an intense icterus, consisting of itching of the surface, depressed spirits, slow action of the heart, muddy urine, and a general yellowness or jaundice.

Course, Duration, and Termination.—The subsequent behavior of cases of hepatic congestion offers wider differences than exist in the clinical history. The cases of congestion due to obstructive diseases of the heart or lungs develop slowly and continue indefinitely, and their course and duration are those of the cardiac or pulmonary disease. In these cases important alterations occur in the liver ultimately ; it undergoes atrophy, obstruction to the portal circulation is added to the stasis in the general venous system, and ascites slowly forms. In the acute cases due to climatic and hygienic causes, the course is short, but the symptoms are violent. The whole duration of such an attack will not be more than a week or ten days, and the termination is in health. The same causes which produce the attack will operate in the future, and other attacks will succeed, and ultimately, in some cases, chronic disease of the liver will be established ; but, if the causes cease, the effects will also. In the nervous cases, the jaundice reaches its maximum in a few hours, and then begins to decline, and usually lasts four or five days, terminating in recovery.

Diagnosis.—The acute form of congestion may be confounded with jaundice from catarrh of the bile-ducts, the symptoms being much the same ; but the duration of the cases differs, and the latter is preceded by symptoms of gastro-duodenal catarrh, while in the former these symptoms succeed to the disturbance in the hepatic functions. The congestion due to obstructive pulmonary or cardiac disease is diagnosticated by its clinical history and the association of the two groups of lesions. The contraction of the liver, which succeeds to enlargement in the cases of nutmeg-liver, may be confounded with cirrhosis ; but, as these states have been confounded by pathologists, the differentiation is not important from the clinical standpoint.

Treatment.—The treatment of the cases due to pulmonary or cardiac obstruction is a question of the management of the lesions, cardiac or pulmonary, as the case may be. Not unfrequently, before the heart and lungs are incommoded in mitral disease, the hepatic functions are so disturbed as to demand attention. The timely prescription of digitalis of barium, or of nitroglycerin, may afford relief, not given by the remedies for disorder of the liver. As the condition is one of abnormal fullness of the venous system of the liver, relief is afforded in those of full habit by leeches around the anus. Unfortunately the need for digitalis, to diminish the leak at the mitral and for leeches to unload the distended veins, continues. Free watery evacuations, produced by salines, are highly useful ; but in the progress of this disease the congestion of the mucous membrane excites a catarrh and diarrhœa, so that the limit of utility by saline purgatives is soon reached. In the acute congestion due to climatic or malarial causes, no remedy is so efficient as a full dose of quinine (grs. xv—℈j) with morphine (gr.¼—½). Small doses frequently repeated may, if preferred, be employed, but the large dose is more efficient. A mild saline laxative, to keep the bowels soluble (the Saratoga waters may be used), is necessary, and elimination by the kidneys should be maintained by the use of lemonade and diluents. Fomentations, turpentine-stupes, etc., applied to the hepatic region are serviceable. When the attacks are due to errors of diet, spirituous liquors, and similar abuses, there must be a change in the habits of the individual. Abstinence, the use of a laxative, and quiet, will effect a cure, provided the excesses have been recent, and alterations of structure have not occurred in the liver.

INTERSTITIAL HEPATITIS—SCLEROSIS OF THE LIVER—CIRRHOSIS.

Definition.—By the term *interstitial hepatitis* is meant an inflammation of the intervening connective tissue. An induration of the organ is the result of this process, and hence it is entitled *sclerosis*, just as this term is used for corresponding states of other organs—as sclerosis of the kidney, sclerosis of the lungs, etc. Cirrhosis is the French term derived from the Greek word *kirros* (yellow), so named on account of the color of the liver; but it is a very inappropriate designation, and should cease to be used.

Forms.—The ordinary form of cirrhosis, and that to which this term is applied, is the contracting, designated the *granular* liver, because of the appearance on section. The nodulated character produced by the new-formed connective tissue in contracting, has given to this affection the familiar English name, *hob-nailed* liver.

There are some other forms characterized by hyperplasia of the connective tissue, and atrophy of the proper gland elements, in which no contraction ensues, the organ remaining permanently enlarged. To these forms is applied the term *hypertrophic cirrhosis*. One of these is supposed to arise spontaneously; another succeeds to inflammation of the ducts, and obstruction caused by hepatic calculi. A third form of the hypertrophic is the *fatty*, the connective tissue undergoing fatty degeneration without contracting.

Causes.—This is a disease of adult life, and rarely occurs before the period of puberty, chiefly because the conditions are wanting at this time. Griffith reports a case in a child of ten; Cayley, in another child of six; and Murchison, in a boy of ten. Nothing definite as regards the cause was known in the first two, notwithstanding a searching investigation; in the other, the abuse of spirits, medicinally and otherwise, was ascertained.* Murchison has never met with an example of hob-nailed liver in which excess in the use of spirits had not been made out. There can be no doubt that the male sex is more frequently attacked than the female, not because there exists any inaptitude in the latter, but because of the difference in habits. The great factor is the free use of alcoholic liquors. The amount which constitutes excess differs in different individuals ; in some subjects a small amount of alcohol, daily, suffices to set up the interstitial inflammation, when another person would not be affected by it in any way. It is highly probable that hereditary syphilis is a cause, but there are obvious difficulties in the way of a correct determination of this point. The form of atrophy which succeeds to the chronic stasis of the liver in obstructive cardiac disease is often confounded with sclerosis proper, but the change begins by an atrophy of the hepatic cells next the intra-lobular vein in the former ; whereas, in the latter, the atrophy begins in the peripheral cells.

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* "Transactions of the Pathological Society," vol. xxvii, 1876, pp. 186, 194, 199.

Sclerosis has been observed to follow impaction by gall-stones and the paludal cachexia.

Pathological Anatomy.—In the first stage, the organ is somewhat increased in size and hyperæmic ; its parenchyma is somewhat denser, by reason of the presence of a viscid, reddish-gray material, which consists of fine connective-tissue elements, containing spindle-shaped cells (Förster).* The development of this material imparts to the parenchyma a granular aspect. The color of the organ is at this period a brownish-red, whence the name cirrhosis, or it may be greenish by staining of the bile-pigment ; or the deposition of fat may give it a pallid appearance. Thus far, there is an actual addition of material to the organ, and it is somewhat increased in size. The next step consists in the contraction of the new connective tissue and induration. The substance of the liver is distinctly harder, and, on section, the knife is resisted as if passing through fibrous tissue. The surface of the organ is unequal, nodulated, and traversed by distinct, thickened bands of connective tissue (whence the English term "hob-nailed"). The line of section presents a granular appearance, due to the contracting of the intervening connective-tissue elements, and the consequent forced elevation of the softer material of the lobules. The peritoneum is opaque, thickened by organized exudation, the results of local peritonitis, and adhesions are formed to the diaphragm, between the liver and gall-bladder, etc. The appearance of the hepatic tissue is due to a hyperplasia of the connective tissue (Glisson's capsule) surrounding and compressing the groups of cells. The cells themselves, where the growth of connective tissue is sufficient to compress them, undergo a change partly fatty, partly pigmented, and in some places amyloid.

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* *Op. cit.*, p. 264.

The abnormal pigmentation is due to compression of the terminal ducts and stasis of the bile. The vessels of the liver are variously damaged. In those parts where the greatest destruction of cells has occurred, the radicles of the portal vein are obstructed, and the radicles of the sub-hepatic are also closed by compression and lose their connection with the capillaries of the portal. The hepatic artery becomes dilated, and supplies the newly formed vessels of the recently developed connective tissue.* The important alterations occurring in the liver lead to secondary disorders of a serious kind. The interruption to the circulation by closure and obliteration of many of the hepatic capillaries—portal and hepatic—necessarily causes stasis in the whole range of the portal system, including the chylopoetic viscera. The formation of bile is impaired, diminished, and at many points entirely suppressed. The glycogenic and urea-forming functions are disordered to the same extent ; consequently the depuration of the blood and the function of digestion, in so far as the presence of bile is necessary to the latter, are hindered or prevented.

HYPERTROPHIC CIRRHOSIS.—In that form of cirrhosis which is entitled *hypertrophic*, the liver continues enlarged, and may weigh from five to eight pounds. It also retains its shape, remains smooth usually, but may be marked by small prominences, due to the compression of islets of parenchyma by the increasing connective tissue. While, in ordinary cirrhosis, the new-formed connective tissue incloses groups of lobules, and is hence designated *annular or multilobular cirrhosis*,† in the hypertrophic form, single lobules are inclosed ; hence the term *monolobular*. The initial change in ordinary cirrhosis begins in the connective tissue about the interlobular vein ; in hypertrophic cirrhosis, about the interlobular duct. In the latter, processes of connective tissue, enlarging, project into the lobule, separating the rows of hepatic cells composing it, and these undergo atrophy. Broad bands of connective tissue newly formed extend through the organ between the parenchyma islets.

As with these changes in the connective tissue, inflammation of the interlobular ducts ensues, with this form of cirrhosis there is more or less jaundice, and hence it has been entitled "biliary cirrhosis." The spleen also enlarges considerably by simple hypertrophy. The other changes in related organs are the same as those of ordinary cirrhosis.

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* Cornil, "Note sur l'état anatomique des canaux biliaires et des vaisseaux sanguins dans la cirrhose du foie," "Bull. de l'Acad. de Méd.," "Gaz. Méd. de Paris," 1873.

† Charcot and Gombault, "Archiv. de Physiol. Normal et Pathol.," 1876, p. 453 *et seq.* Also, Charcot, "Leçons sur les Maladies du Foie," etc.

Symptoms.—The initial symptoms are those of congestion—some heaviness, and dragging in the right side, and increase in volume, the liver projecting a finger's breadth below the ribs. There will be present, usually, some pain and tenderness on pressure, and now and then acute pain with a febrile movement indicative of local peritonitis. A slight icterode hue of the skin may also appear, and rarely jaundice. Again, in other cases, before symptoms referable to the liver manifest themselves, gastro-intestinal disorders—gastro-intestinal catarrh—occur. The appetite is poor, and food occasions distress; there is acidity, and acid matters are regurgitated; often in the morning there are much nausea and great straining, some acid, glairy mucus and bilious matter coming up after much effort. The bowels are sometimes relaxed, sometimes constipated, and now and then blackish, tar-like, semi-solid discharges occur. As intestinal hyperæmia is always present, and sero-mucus constantly poured out, diarrhœa soon comes to be the usual condition. A troublesome meteorism is a constant symptom, and this is due to decomposition of certain foods and a paretic state of the bowels. There are also cases, but rarely, in which the development of sclerosis takes place silently, and the first symptom to awaken attention is ascites. As respects size, the liver usually enlarges at first, but contraction soon comes on, and a considerable reduction takes place, the area of hepatic dullness being correspondingly reduced, except in that form of the disease known as hypertrophic cirrhosis, in which the dullness on percussion increases in all the diameters, or at least does not diminish. As the splenic forms a part of the portal system of veins, a constant stasis is maintained in the circulation of the spleen, and hence this organ remains swollen; but there are variations in its size, due to the formation of a collateral circulation, and occasionally to the development of a sclerosis in the organ. A constant stasis is also maintained in the intestinal mucous membrane, with the results already mentioned. An attempt at compensation for the obstruction in the venous system of the abdomen is made by enlargement of certain communicating veins, which in health are but slightly auxiliary to the regular route of communication. On the surface of the abdomen, from the xiphoid appendix to the pubis, veins appear, which were previously invisible; they are the communicating veinules between the epigastric and internal mammary, forming an irregular, feather-shaped figure; interlacing vessels also develop along the rectus muscle, laterally; communication is established between the parietal veins and the accessory vena porta of Sappey, and those branches of this accessory portal, communicating with the epigastric and internal mammary veins, form a cushion, bluish in color, of distended vessels around the umbilicus (caput Medusæ): communication also takes place between the inferior mesenteric and the hypogastric veins, through the hæmorrhoidal, and between the anastomoses of the portal with the œsophageal and diaphragmatic veins.

Hæmorrhages result from the stasis—hæmatemesis or vomiting of blood, and intestinal hæmorrhage; the vessels yield under the increased pressure; or thromboses form in the stomach-veins, solution of the affected mucous membrane occurs, and an ulcer is the result. The author has seen two cases of cirrhosis in which frequently recurring hæmatemesis caused death, the hæmorrhage coming from small ulcers in the vicinity of the pylorus. The black, tar-like stools which are passed now and then in contracted liver consist of blood altered by the intestinal juices. The same obstruction of the portal circulation leads to the formation of hæmorrhoids, which often bleed freely and thus afford relief. Besides the interference with the digestive function due to the gastro-intestinal catarrh, the solution and absorption of certain kinds of food are prevented by the absence of the bile. These are especially the fatty and saccharine matters, and bile has the peculiar property of aiding the absorption of fats. Further, it plays the part of an antiseptic agent, and prevents the decomposition of food in the small intestine: when bile is absent the fæces are not only wanting in the proper color, but they have a peculiarly fetid odor—the odor of decomposition—and the gas passed has the same foul smell. A gradual emaciation is the necessary result of this morbid condition of the intestinal digestion. The integument of the face, neck, and forearms acquires a peculiar, earthy, icteroid hue, but a real jaundice is not common in cases of sclerosis. Sometimes with the first congestion, which initiates the morbid process, jaundice is a symptom, but it soon disappears and the earthy, fawn color, so characteristic in these cases, gradually develops. In those cases of sclerosis succeeding to impaction by gall-stones, jaundice has been a prominent symptom. When the cells have atrophied, and the canaliculi are obliterated, resorption of bile is no longer possible. The very considerable interference with the process of digestion produced by sclerosis and the retention in the blood of those effete materials which it is the function of the liver to remove induce an unhealthy condition of that fluid, and hence venous stigmata appear on the face and nose, and bleeding occurs from the nose, lungs, peritoneum (peritonitis hæmorrhagica), and elsewhere.* The urine is small in quantity, high colored, brownish, deficient in urea, but loaded with urates which are deposited in great abundance along with much coloring matter. Œdema of the feet and ankles succeeds to ascites, and the genitalia become much swollen. But the clinical history and treatment of ascites have been sufficiently discussed.

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* "Thèse de Paris," 1874, Azmi Ahmed, "Des hémorrhagies dans la cirrhose."

Course, Duration, and Termination.—The course of interstitial hepatitis is essentially chronic. The first stage, or period of congestion and enlargement, often escapes notice, and only the stage of contraction, with its accompanying derangements, comes under observation. The duration is not fixed, and the termination is governed by the extent of the contraction and the consequent interference with function, but especially by the existence or appearance of such complications as mitral disease, emphysema of the lungs, and chronic interstitial nephritis. Fibroid change, such as occurs in sclerosis of the liver, may manifest itself simultaneously in other organs, as fibroid lung, fibroid heart, fibroid kidney. Obviously, the course and duration of the hepatic disease will be much influenced by the coexistence of this form of degeneration in other organs. Toward the end of some cases, brain symptoms arise which were at one time supposed to have the same relation to retention of effete products removed by the liver in the normal condition as the cerebral symptoms in albuminuria had to the failure of kidney excretion. By Flint this toxic material is supposed to be cholesterine, and hence the term cholesteræmia which he applies to these cerebral symptoms. This condition of the brain takes the form of stupor, and low-muttering delirium, passing into deep coma. In a few cases sopor and gradually deepening stupor come on early. These mental symptoms are, however, mixed up with the perturbation due to alcoholic excess, so that it is impossible to assign to each factor its proper influence in the development of this state. A large proportion of cases end before these mental symptoms are reached, cut off by intercurrent maladies, such as pleuritis, pericarditis, pneumonia, etc., or die exhausted by hæmorrhage. Some cases proceed to a typical ending by gradual failure, worn out by the difficult breathing from excessive accumulation of fluid, the constant upright position, the ulcerated legs, the bleeding hæmorrhoids, repeated tapping, stupor, delirium, and gradually deepening coma.

Diagnosis.—When all the usual symptoms of sclerosis are present, and the subject of them has been given to alcoholic intoxication, there can be no difficulty in coming to a diagnosis by exclusion. Furthermore, sclerosis is greatly more frequent than any of the diseases with which it may be confounded. The difficulties of differentiation occur with pylephlebitis, fatty liver, hydatid cysts, cancer or tuberculosis of the peritoneum. In pylephlebitis or inflammation with thrombosis of the portal vein, there may be present the same symptoms as in sclerosis, but they arise suddenly, and are not preceded by the symptoms of congestion and a history of alcoholic abuse. Fatty liver is one of the complications of phthisis, and also occurs in the obese, or in those having the tendency to obesity and who eat and drink freely and lead sedentary lives. Although the symptoms referable to the liver are similar to those which are present in sclerosis, there are important points of difference. In fatty liver emaciation is wanting; the organ is enlarged and smooth, instead of being contracted and nodulated. In hydatid cyst, there is a slow, gradual, and painless enlargement, with but little interference in the function of the liver, and without the secondary gastro-intestinal disorders. On palpation, a large, soft, elastic growth can be made out, and having that peculiar symptom, the "purring tremor." These symptoms are all wanting in sclerosis. Cancer differs from sclerosis in that the pain is greater, the wasting more rapid, the liver presents large protuberances, and secondary deposits in the mesentery can be felt in advanced cases. Cancer and tubercle of the peritoneum are accompanied by symptoms much like sclerosis. They may be differentiated by attention to the following points: In sclerosis, there is enlarged spleen; the urine is deficient in urea but contains leucin and tyrosin, and casts an abundant deposit of urates and coloring matter; in cancer or tubercle, the spleen is not enlarged; the urine contains its proper proportion of urea, and is pale and watery. In cancer or tubercle of the peritoneum, there is great tenderness of the abdomen; the ascites develops quickly; the strength and flesh rapidly decline, and there are usually cancer or tubercle deposits in other organs.

Prognosis.—The course of sclerosis is usually continuously downward, and hence the prognosis is unfavorable. The author believes that the opinious as to its incurability, based on experience, must be somewhat modified now, in view of the results of modern treatment.

Treatment.—At the outset the author must condemn the use of mercurials given with a view to correct the hepatic secretions. The secretory function is disturbed, because the liver-cells have atrophied and the ducts are closed. When this result is reached, no treatment can modify the case, for remedies can not restore lost parts. Before important changes have occurred, although new connective tissue has formed, and some contraction has taken place, the author believes that much may be done to arrest the morbid process. There is a group of remedies which have a selective action on the liver, the metals chiefly: gold, silver, copper, arsenic, mercury, and phosphorus, which have the property of improving the nutrition of the liver if used in a small quantity for a long period. The most efficient of these are the chlorides of gold and sodium, the corrosive chloride of mercury, Fowler's solution, and phosphorus in the form of phosphites or phosphates. When there is much irritability of the gastro-intestinal mucous membrane, two drops of Fowler's solution, with two to five drops of opium tincture, three times a day, will be most easily borne. If there is less irritability, the chloride of gold and sodium ($\frac{1}{20}$–$\frac{1}{15}$ gr.), or corrosive chloride of mercury ($\frac{1}{20}$–$\frac{1}{60}$ gr.), *ter in die*, can be administered. No good result should be expected unless the remedies are kept up for several months. The author has seen surprising results by the long-continued use of sodium phosphate in these cases—given in ℈j—ʒj doses three times a day. The good effects of both remedies may be obtained by joint administration—the phosphate in solution, the chloride in pill form. When it is considered desirable to give phosphates and arsenic together, phosphate of soda and arseniate of soda may be combined. If there is a suspicion of syphilitic taint, the iodides of potassium and ammonium and the bichloride of mercury are the appropriate medicaments. The mineral acids, which at one time were supposed to be efficacious in the treatment of this hepatic disorder, are now rarely employed, except to facilitate digestion. The nitro-muriatic bath is a serviceable topical application, especially the general bath, to improve the condition of the skin, which is dry, harsh, and scurfy. Attention to the diet is of the first consequence. Fats and saccharine foods, not undergoing solution and absorption, decompose and add to the existing mischief. The continued use of skimmed milk freely is a dietectic measure of the highest importance. Those components of a diet convertible into peptones should be directed, and the most easily digested substances only. When ascites forms, it must be treated according to the principles already set forth under that head ; the activity of the kidneys must be maintained, and puncture practiced according to necessity.

SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER.

Definition.—The hepatitis which terminates in suppuration is localized to a special part, and the rest of the organ, outside the area of suppuration, continues comparatively normal. It is parenchymatous inflammation in that the proper structure of the organ—the gland-cells—is the seat of the inflammatory process. It is a suppurative hepatitis, in that the tendency is to the formation of matter, and the resulting abscess is the special feature demanding attention. A well-founded distinction is made between pyæmic and tropical abscesses—the former, a result of blood-poisoning ; the latter, caused by inflammation of the liver. It is the latter form which is intended by the term suppurative hepatitis, but the *post-mortem* changes and the clinical history, so far as the liver itself is concerned, are the same in the two forms.

Causes.—External injury but rarely excites suppurative inflammation, and a blow on the right hypochondrium will more frequently cause an inflammation of the hepatic peritoneum than of the hepatic substance. Blows are more apt to cause abscess of the liver in warm than in cold countries. Climate is one of the principal factors.* A warm climate, an alluvial soil, and miasmatic influences, are more influential in combination than climate alone. Abscess of the liver is very common in the great interior valley of North America—along the Mississippi and its tributaries, within the malarial area—as it is in India, and because of the same etiologic and climatic conditions. Without producing the objective phenomena of fever, malaria disturbs the hepatic functions, but the disturbance is still more decided when the poison is intense enough to cause fever. Dysentery and ulceration of the intestines have so frequently coincided in appearance with, or have preceded, abscess of the liver, that a causal relation is supposed by many to exist between them. In the interior valley of this continent, at Cincinnati, the author saw many cases which had succeeded to attacks of malarial fever, and to dysentery—especially proctitis—the lesions of which are situated chiefly or wholly in the rectum. Frerichs,* Murchison,† and some other systematic writers, after a thorough examination, maintain the opposite view, that the supposed relation between abscess of the liver and dysentery is merely coincident, and is not causal. Waring's ‡ statistics seem quite conclusive against the view that such a relation exists: thus, "out of 2,758 cases of dysentery treated in the Madras Presidency, abscess of the liver occurred 68 times, being in the proportion of 2½ per cent. nearly." In the same author's 300 cases of abscess of the liver, "hepatitis was the primary affection in 131, or 43 per cent., while only 82, or 27 per cent., were admissions from dysentery." Budd § holds that a poison generated in the intestine by the decomposition of materials from ulcerations is the chief factor in the causation of abscess. Moxon ‖ also maintains that "almost all tropical abscesses are secondary to dysenteric or other ulcerations, and that primary abscess of the liver is at least as doubtful as primary suppuration of the brain." The concurrence of hepatic abscess and dysentery is too frequent not to be related in some way; it is clear that many, but probably not a majority, of the cases thus originate, and, when so caused, the abscesses are pyæmic, multiple, and secondary. Large abscesses of this kind are due to the coalescence of neighboring smaller ones. A very intimate causal relation exists between ulceration of the cæcum or of the appendix vermiformis and abscess of the liver, numerous cases of the kind having been reported.¶ A large number are doubtless due to hepatitis—the so-called tropical abscesses. A variety of causes are concerned in the production of others. The habits of individuals are not without influence, especially the use of stimulants, highly seasoned dishes, condiments, etc. Suppuration has been caused by the impaction of calculi, by the lodgment of a lumbricoid worm, etc. It is a more common malady in men than in women, and from the twentieth to the thirty-fifth year. A case is reported by Grainger-Stewart, in which abscess of the liver followed dilatation by the bile-ducts.*

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* Sachs, "Ueber die Hepatitis der heissen Länder," Berlin, 1876. Separat-Abdruck aus von Langenbeck's "Archiv," Band xix.

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* "Diseases of the Liver." Translated by Murchison. Syd. Soc., vol. ii, p. 108.

† "Clinical Lectures on Diseases of the Liver," etc. Second edition, p. 177.

‡ "An Enquiry into the Statistics and Pathology of Some Points connected with Abscess of the Liver, as met with in the East Indies." By Edward John Waring. Trevandrum, 1854.

§ "On the Diseases of the Liver," p. 83, *et seq.*

‖ "Transactions of the Pathological Society of London," vol. xxiv, p. 116, 1873.

¶ Ibid., various volumes.

Pathological Anatomy.—That a certain proportion of cases of hepatic abscess are due to embolic deposits, coincident ulcerations existing in the intestine, is probably true, but the facts of observation which support this theory are surprisingly few. Frerichs † reports one of embolic blocking of a vessel at the site of a commencing abscess, and a few others have been recorded. Förster ‡ holds that a miasmatic infection of the blood is caused by the ulceration in the intestine. Ulceration of the cæcum is more frequently the source of embolic abscess of the liver, and it is probably this state of things which explains the relatively numerous examples of single abscess found in this connection. The cells become cloudy and granular by the presence of an albuminous matter deposited in them. Liebermeister maintains, but he is alone in this opinion, that the initial change is in the connective tissue ; but Rokitansky, Virchow, Frerichs, Förster, and others, refer the first changes to the cells of the hepatic parenchyma, and the alterations in the connective tissue to a subsequent period.

Those parts of the hepatic parenchyma in which the liver-cells are undergoing disintegration, at first have a reddish-yellow appearance, and at some points contain patches of pigment of a bright yellow color, and are surrounded by a translucent pale-gray ring. The acini, the seat of this process, are distinctly enlarged, become softer, and disintegrate. The center of each inflamed patch early becomes yellow, which indicates the beginning of suppuration. The size of these points of suppuration is at first small, but those in close proximity coalesce, forming an abscess—a purulent collection. These abscesses are filled with pale-yellow pus, and the borders of the collection consist of dark-red, disintegrating gland-tissue, projecting in the form of softening shreds into the purulent depot. They vary in size from a pea to a hen's egg, or may attain much larger dimensions. Important changes take place in these purulent collections as they grow older : the walls become smooth, and are lined by connective tissue, the pus thus becoming encysted, or absorption occurs, the walls of the abscess approximate, unite, and ultimately nothing remains but a linear cicatrix. So perfectly does repair go on and is completed, that in some years afterward scarcely a trace of the original mischief can be detected. In other cases no limiting membrane is produced, the inflammation extends, and an enormous purulent collection, which tends to external discharge in some direction, is formed, and enlarges by continual accessions of purulent matter. It does not often happen that such a collection bursts into the peritoneal cavity, exciting fatal peritonitis, but it tends to perforate the abdominal wall, or dissects downward along the spine, discharging in the inguinal region or by the sacrum posteriorly, or it ulcerates through into the stomach, duodenum, or colon, or makes its way upward, perforates the diaphragm, the lungs, and is discharged through the bronchi. These abscesses have also entered the vena cava (case of Colin *), have ulcerated into the pericardium, etc., but such accidents are comparatively rare.

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* T. Grainger-Stewart, "The Edinburgh Medical Journal," January, 1873.

† "Diseases of the Liver," *op. cit.*

‡ "Lehrbuch der pathologischen Anatomie von Dr. August Förster." By Dr. Siebert. Jena, 1873, p. 267.

The size of an abscess of the liver varies from an ounce or two to a gallon. In 69 cases in which this point was noted, 16 contained one to two pints, and 12 two to three pints ; and these may be regarded as of the usual sizes. As respects limitation by a neo-membrane, the cases are not numerous in which definite statements are made ; in 53 the abscesses were encysted in 36 and not limited in 17, but it is doubtful if this relation exists throughout a large number of unselected cases. Of Waring's 300 cases, 169, or somewhat more than one half, remained intact ; of the remainder, much the largest number of the spontaneous discharges occurred by the thoracic cavity—42—and of these 28 occurred through the right lung. As respects the lobe of the liver, which is usually the seat of the abscess, the statistics of various observers agree. Selecting Waring's 300 cases for exemplification, we find that the purulent collection was in the right lobe, alone, in 163, and in both right and left in 35. The number of abscesses present at the same time is influenced greatly by the cause ; in the pyæmic, there may be a dozen or more ; in the other form, from one to three usually. Although fetid decomposition is not uncommon,† yet true gangrene is very rare.

Symptoms.—Notwithstanding the importance of the organ, abscess of the liver of considerable size may exist without there being any local or systemic symptoms to indicate its presence. These latent cases occur in the course of chronic dysentery and pyæmia, and fail of recognition because masked by existing symptoms, or they are latent because the inflammation occurred in the deepest part of the right lobe, and did not involve the peritoneum, nor did the abscess compress the bile-ducts, and was limited by a neo-membrane. A typical case following a recognized injury, or due to impaction of calculi, will present characteristic symptoms, and the diagnosis will be easy, but many other cases may not only be difficult of recognition, but in some a diagnosis will not be possible.

The onset is marked by the phenomena which attend an inflammatory affection ; a chill, or chilliness, aching of the back and limbs, headache, a dry skin, a coated tongue, bilious vomiting, increased action of the heart, a rise in the arterial tension, are the systemic symptoms. Locally, there is a feeling of uneasiness, constriction, weight, dragging, and often considerable pain and tenderness, especially if the hepatic peritoneum is involved. In some cases a pain is felt in the top of the shoulder—a tensive pain—and it is experienced in the right shoulder when the right lobe is affected, and in the left shoulder if the left lobe is the seat of mischief, and in some cases in both simultaneously. Its value as a symptom is not great, for it is present in other hepatic diseases, and may be a merely rheumatic or neuralgic pain. On palpation and mensuration, an increase in the size and density of the liver can usually, but not invariably, be made out. The area of hepatic dullness is increased in all directions, and may be considerably so if the purulent collection is a large one. Pushing up the diaphragm and displacing the lung, the area of dullness and the absence of voice and breath sounds may extend up to the fourth, to even the lower margin of the third rib, and downward several finger-breadths below the margin of the false ribs, furnishing all the signs of hydropneumothorax.[*] Jaundice is present in less than one third of the cases, and then varies much in intensity, but it is general, and the urine is loaded with bile-pigment, and, when the liver is much damaged, contains leucin and tyrosin instead of urea. Jaundice appears early in those cases of abscess due to the impaction of calculi—soon after or with the initial symptoms, which are those of hepatic colic—and much later in those which are the usual cases, due to the pressure, on the hepatic duct, of the abscess. When pus forms there is usually a decided rigor, and these shiverings recur irregularly, and are followed by fever and sweats. Like the other characteristic symptoms, these are often entirely absent. The fever, chills, and sweats are much more pronounced in the so-called pyæmic abscesses than in those arising from hepatitis. The irritability of the stomach is enhanced by the occurrence of suppuration ; the frequency and persistence of the vomiting at this period is an important indication, much insisted on by Maclean[†] and Fayrer.[‡] The vomiting may have the bilious character, with a large evacuation of bile, and the alvine dejections may have the same character ; the vomit may consist of watery mucus, and, rarely, of blood. There will be an increase of the dysenteric symptoms, if this disease had been in existence when the abscess formed, or diarrhœa or dysentery may occur when suppuration takes place. The size of the liver lessens somewhat, and the area of hepatic dullness diminishes when pus forms, if the abscess be inclosed; but, if no limiting membrane is formed, the dimensions of the organ gradually enlarge. The diminution in size is maintained, and a gradual return to the normal is the rule, when the pus is absorbed and the cavity cicatrizes. Fluctuation is felt and can be detected only when the purulent collection attains to great dimensions. If the abscess tends to spontaneous recovery by absorption, or after discharge of pus, the local pain and tenderness subside, the pulse falls to the normal, the stomach is no longer irritable, appetite returns, and digestion is resumed. If, however, the abscess enlarges, the distress in the hepatic region and the tenderness increase; movements, especially of breathing and coughing, awaken deep-seated soreness and pain; breathing becomes difficult by pressure on the lungs; the heart is sometimes displaced upward and to the left, which adds to the existing præcordial uneasiness and to the difficulty of breathing; and a harassing and painful short, dry cough, induced by irritation of the pneumogastric and phrenic nerve-filaments, adds greatly to the distress. As a tendency to discharge through the right lung exists in a large proportion of cases, the base of this lung and the neighboring pleura are affected by a localized pleuro-pneumonic process, with the usual physical and rational signs of that complication. Adhesion of the pleural surfaces takes place, and a channel is formed communicating with a bronchus, through which discharge occurs. Less often a secondary suppurating cavity is constructed by the pleural adhesions. Rarely the pericardium is opened, and death caused by sudden distention of the sac with pus. If rupture takes place into the peritoneal cavity, this untoward accident is announced by sudden, intense pain and collapse; if into the intestine, purulent and bloody evacuations indicate it, while lessened size of the liver and less tension and pain also coincide; if the pus dissects outwardly through the hypochondrium, a large, puffy, and fluctuating tumor forms.

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* "Gazette Hebdomadaire de Méd. et de Chir.," No. 33, 1872.

† Rigal, "L'Union Méd.," No. 134, 1873.

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[*] Rigal, "L'Union Méd.," No. 134, 1873.

[†] "The Diagnostic Value of Uncontrollable Vomiting." Dr. W. C. Maclean, "British Medical Journal," August 1, 1873.

[‡] Sir Joseph Fayrer, ibid., September 26, 1873.

The variations in the symptoms of hepatic abscess are very remarkable. There may be no local symptoms—no pain, no tenderness, no enlargement. When the purulent collection tends downward below the ribs, there may be fluctuation, and when it has attained to great dimensions; but it is a comparatively rare symptom. In much the largest number of cases, the pus forms in the upper and superior part of the right lobe, in a situation where fluctuation can not be developed. Pain may be entirely absent: in Waring's 300 cases of hepatic abscess, pain was not present in 20. The reflex shoulder-pain is much less constantly experienced; it is more frequently wanting than it is felt. Gastric derangement of any kind may not exist, and the patient may have a good appetite. The importance of severe vomiting as a symptom of suppuration is not impaired by the fact that exceptional cases are encountered, but vomiting and severe and uncontrollable vomiting are highly significant, andvery rarelyabsent. Vomiting is increased by extension of disease to the peritoneum, and by pressure of an enlarging abscess directly upon the stomach. Although the bowels may be undisturbed in exceptional cases, dysentery is present in a considerable proportion—according to Waring, in 82 in 300 cases—but dysentery sometimes succeeds to the abscess, and is apparently caused by it. Ascites occasionally occurs when the abscess compresses the portal, and jaundice usually accompanies it, for the common or hepatic duct is encroached on at the same time.

Course, Duration, and Termination.—So much obscurity exists in regard to the initial symptoms, so much variation in the behavior of cases, that no defined course can be laid down. The duration is equally uncertain and irregular. A typical case without complication may pass through its several stages in about seventy days if the pus is discharged by a favorable channel; if the pus undergoes absorption, and the cavity closes by cicatrization, several weeks longer will be necessary. The initial symptoms will occupy less than a week, for suppuration appears in a short time after the hyperæmia, and the breaking down of the hepatic tissue proceeds rapidly, so that an abscess of considerable size will form in seven to ten days. Then comes on a period of septicæmic fever —remittent in type, with irregular sweats, in the acute cases with abscess of large size, and intermittent with long periods of freedom from fever in the subacute and chronic cases, with abscess of moderate size. The course of abscess of the liver is much affected by the development of a limiting neo-membrane. When this membrane is formed, if no complications are present, there may be a "latent period" of considerable duration—a period characterized by the absence of local and systemic symptoms. This quiescent state may continue several weeks, months even ; then acute symptoms arise, which are often misinterpreted, and supposed to be the initial symptoms, and the abscess formed, the product of the recent disturbance. If, on the other hand, there is no limiting membrane formed, and the suppuration extends, the septicæmic fever persists, and the patient sinks into a typhoid state, with low-muttering delirium, and death from exhaustion.

Cases of acute abscess without complication, discharging in a favorable direction, recover with considerable promptitude. Early and successful use of the aspirator for the evacuation of pus shortens the duration of a case materially. Convalescence is very tedious when fistulous communication exists through the lungs, the parietes of the abdomen, and elsewhere. The author has met a case of fistula of the right hypochondrium discharging somewhat after eighteen months. During the existence of such purulent formation and discharge, night-sweats, diarrhœa or dysentery, a poor appetite, and feeble digestion combine to maintain a condition of debility for a long time, or there may be a continuous, gradual failure, terminating in exhaustion and death. In the acute cases which terminate fatally there are usually intense hectic, profuse sweats, uncontrollable vomiting, and rapid failure of the vital powers. The cases associated with dysentery are very protracted and very fatal; they rarely cicatrize, and less frequently discharge externally than do the uncomplicated cases (Frerichs). The condition of patients who recover is not always that of health. Very often the intestinal digestion is impaired because of the insufficient supply of bile, and the functions of the stomach and intestines are interfered with by adhesions and contracting bands of lymph which limit the movements of these organs and narrow their capacity, or obstruct the passage of their contents.

Prognosis.—How favorable soever may be the apparent condition in any case of hepatic abscess, the prognosis must be guarded, for unexpected complications may arise, and the known dangers are uncertain in their behavior. The pyæmic abscesses are more numerous, are due to a poisoned state of the blood, and are always fatal. The direction taken by the abscess is an important element in coming to a conclusion; discharge by the lungs is most favorable; by the external integument the next, and by the intestinal canal, third. Early evacuation by the aspirator lessens materially the dangers and must enter into the question of prognosis. In eighty-one cases of hepatic abscess evacuated by operation, collected by Waring, there were fifteen recoveries—18·5 per cent. In McConnell's,* fourteen cases in which the aspirator was used, six died and eight recovered—fifty-seven per cent. Both sets of statistics were gathered in India, but the former were cases which occurred before 1850, and the latter since the aspirator came into use. Of twenty-five cases of recovery without interference, also by Waring, there were ten in which the matter was discharged through the lungs, and seven by stool. The size of the abscess, its position, the condition of the patient in respect to digestion and nutrition, and especially the presence or absence of complications, are elements which must be taken into consideration in coming to conclusions.

Diagnosis.—Hepatic abscess may be confounded with echinococcus of the liver, dropsy of the gall-bladder, scirrhus, abscess of the abdominal wall, effusions, especially purulent, into the right thoracic cavity, etc.

A tumor or enlargement formed by echinococci is unaccompanied by pain or tenderness, the growth is slow and without constitutional disturbance, when palpated is elastic, fluctuating, and furnishes that most characteristic sensation, "the purring tremor." An abscess of such a size would be accompanied by pain, tenderness on pressure, by septicæmic fever, at least frequently; there would be wasting and diarrhœa, often severe vomiting, and the sense of fluctuation would be free from purring tremor. The very important aid to diagnosis afforded by the exploring trocar should not be neglected, and its indications may indeed be decisive. The fluid of an abscess is purulent, and, if hepatic, contains portions of the tissue of the liver;* if of a hydatid cyst, a straw-colored, serous fluid, containing the characteristic echinococcus hooklets. An enlarged gall-bladder is a pyriform tumor of variable size, elastic and fluctuating when its contents are fluid, or hard and nodular when enlarged by calculi. When the accumulation is a product of the metamorphosis of bile and mucus, the growth is very slow, and the symptoms *nil*—a very different history from that of abscess; on the other hand, a purulent fluid forming, will be accompanied by hectic, sweats, emaciation, etc., and a differentiation is not possible. In cases of this kind there has been a history of attacks of hepatic colic; the last one having determined the series by a closure of the cystic duct. Abscesses of the abdominal wall of large size, and situated in the right hypochondrium, may be very confusing, but the distinction may be made by the history, which does not include any disturbance in the hepatic functions, and has not been preceded by any symptoms of disease of any kind. The history begins with the formation of a tumor in the hypochondrium. The most certain means of diagnosticating consists in the microscopic examination of the purulent matter, and in determining by the passage of the aspirator needle that the pus is contained in an abscess exterior to the ribs. It is impossible to decide between an hepatic abscess and an abscess formed between the hepatic and parietal peritoneum, which may be the result of a local peritonitis, or of an hydatid cyst undergoing destruction by suppuration. Multiple abscess of the liver has been mistaken for cancer of the stomach.† The pain, vomiting, wasting, may mislead, but the marked difference in the history of the two affections, as well as the local symptoms, ought to prevent such an error. The most difficult problem in the diagnosis of hepatic abscess is the distinction between abscess and empyema, or hydrothorax. Besides the evidence of the accumulation of fluid filling in the space from the diaphragm to the fourth, even to the third rib, there are almost always present the symptoms of a pneumonia in preparation for the evacuation by the lung. The physical signs will be the same, but the history of the case will exhibit important differences: in the one case the accumulation of fluid will have been preceded by the signs and symptoms of pleurisy or pleuro-pneumonia; in the other, by the signs and

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* Remarks on pneumatic aspiration with cases of abscess of the liver treated by this method. "Indian Annals of Medical Science," July, 1872.

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[*] Dr. Samuel Fenwick, "Lancet," November 17, 1877, "On the Detection of Particles of Hepatic Structure in Abscess of the Liver." The pus is shaken up with some distilled water and put aside in a conical wineglass. When settled, it is examined with the microscope, or it is shaken up with some distilled water to which a few drops of ammonia have been added, and then, after subsidence, examined.

[†] Dr. W. Crumb, "Philadelphia Medical and Surgical Reporter," March 14, 1873.

symptoms of hepatic inflammation. Here, again, the aspirator may be invoked to make the diagnosis clear—the presence or absence of bits of hepatic tissue will prove the abscess to involve, or not, the liver-substance.

Treatment.—As suppuration occurs so promptly after the initial hyperæmia, it is doubtful whether any effort to prevent the formation of pus can be successful, but the extension of the area may be checked or limited. As soon as the symptoms manifest themselves, a large dose of quinia (twenty grains) should be given at once, and decided cinchonism be maintained by the same dose at proper intervals, or by smaller doses more frequently. That quinia has the power to check the migration of the white corpuscles is well established, but it is equally true that large doses are necessary to accomplish this. Morphia should be combined with it, unless some contraindication exist, and especially if there be much pain and the peritoneum be involved. Warm fomentations and turpentine-stupes should be applied over the right hypochondrium. At the earliest moment when the existence of pus can be made out, or there are good reasons to suspect its presence, an exploratory puncture with the aspirator should be made. The recent experiences of Cameron,[*] Condon,[†] and Sachs[‡] have demonstrated that when the pus can be reached and evacuated a very large proportion of cases recover immediately. It is a remarkable fact that many cases in which the symptoms of abscess exist, and yet no pus is found, are greatly benefited by the puncture. The modern experiences have demonstrated also that, penetrated by suitable needles, no injury is done to the liver, and that repair takes place so perfectly that after death no trace of the operation is visible. The necessity for early evacuation of the pus consists in this, that only a portion of these abscesses are confined by a limiting membrane, and that those thus restricted do not long remain encapsulated, but tend to make their way externally. In Condon's collection of cases there were eight of abscess evacuated by the trocar, of which four recovered, and three of hepatitis, without suppuration, in which the trocar was inserted deeply in the right lobe, all of which were much relieved by the puncture and promptly cured. In Sachs's collection of twenty-one cases there were eight recoveries after puncture—being in the proportion of thirty-eight per cent. Under the old system of using the knife or trocar, when the pus was already pointing, as represented in the statistics of Waring, there were sixty-six deaths in eighty-one cases, making the percentage of recoveries 18·5. When the abscess is large, and repeated punctures are necessary, the author has had excellent results from the injection of tincture of iodine ; it lessens the formation of matter and prevents its decomposition. Mercury was formerly much used in all hepatic affections, but that it is injurious in abscess is now disputed by no one. It is probable that the sulphides, so much and successfully employed in external suppuration, will be found adapted to the treatment of hepatic abscess. The sulphides of sodium and calcium and the sulphurous mineral waters are suitable agents to be so exhibited. As the vital resources of the patient are severely strained, the strength should be carefully husbanded from the beginning. The diet must be generous, and stimulants judiciously administered. When suppuration has occurred, the alcoholic stimulants must be given freely. For the dysentery present in so many cases, ipecac is the best remedy, if prescribed in the necessary quantity—℈j every three or four hours. If there are present old ulcerations of the intestinal tract, copper sulphate is an efficient remedy ; but usually the astringents in turn will be administered in vain.

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[*] "The London Lancet," 1863, June 6th and 13th—"On the Treatment of Acute Hepatitis in its Suppurative Stage."

[†] Ibid., August, 1877, Dr. E. H. Condon—"On the Use of the Aspirator in Hepatic Abscess."

[‡] "Ueber die Hepatitis der heissen Länder," etc., von Dr. Sachs in Cairo, *op. cit.*

GENERAL PARENCHYMATOUS HEPATITIS—ACUTE YELLOW ATROPHY.

Definition.—As the hepatitis terminating in suppuration is confined to a part of the liver, it has been designated Local Parenchymatous Hepatitis, while the term General Parenchymatous Hepatitis is applied to Acute Yellow Atrophy, which consists in an acute diffused inflammation involving the whole organ, and terminating in atrophy. Various names have been applied to this disease, as "malignant jaundice," "typhoid icterus," "hæmorrhagic icterus," etc.

Causes.—Various theories have been proposed to account for the origin of acute yellow atrophy. It has been referred to an excess in the production of bile, to stasis of the bile, to sudden saturation of the hepatic cells with biliary matters contained in the blood of the portal vein. Budd supposes it to be caused by some special blood-poison of unknown nature, which acts especially on the liver. These hypotheses are without facts to support them. That it is an acute, diffuse, parenchymatous inflammation is established by the most recent investigations, but the exciting cause of this inflammation remains unknown. That it is in the nature of a specific morbid poison seems probable, since other organs are simultaneously attacked. It may be that the circumstances are such as to cause the formation of a peculiar *ptomaine*. There are certain points in the etiology of the disease, however, which are well known ; it occurs most frequently in the female sex, and during the state of pregnancy. According to the statistics of Frerichs, in thirty-one cases of this disease twenty-two were females, and one half of these were attacked during the state of pregnancy. It occurs from the third to the sixth month of pregnancy, and in comparatively young subjects, under forty, and rarely indeed after thirty years of age. Other causes have been supposed to exert an influence in its production: as anger—a violent passion having been the apparent cause in cases reported by the older writers—venereal excesses, syphilitic infection, and local miasms. Acute atrophy of the liver has been induced by the changes resulting from typhus fever. A condition analogous to it is brought about by the action of phosphorus, arsenic, antimony, and certain other minerals, and a similar state has been induced by subacute alcoholismus (Rendu).

Pathological Anatomy.—The liver presents a most characteristic appearance—it is much smaller, flattens out by its own weight, is soft so that it tears easily, and has a uniform yellow color. The peritoneal layer is roughened and wrinkled. On microscopical examination, the changes seen are those due to interstitial and parenchymatous exudation. There is, at first, an hyperæmia, traces of which are discoverable at various points, the rest of the organ being anæmic, a result of the subsequent atrophy and obliteration of vessels. Between the lobules there is deposited a grayish-yellow material, which widens the interlobular space, and in those cells which are still recognizable is contained a quantity of an albuminous and fatty matter mixed with pigment.* In the place of the disintegrated cells there is formed a quantity of brownish, fatty granular matter; fat-globules; pigment; bacterian colonies,† and needles of tyrosin and leucin. The ultimate radicles of the portal system and the hepatic artery are obstructed or obliterated. The kidneys also undergo characteristic changes, especially in the cases occurring in pregnancy. The organs are thoroughly stained by the icteric urine, especially the endothelium of the tubules, and besides the cells of the endothelium have become infiltrated by a granular albuminous matter, and are undergoing fatty degeneration. The urine is heavily loaded with bile-pigment, and usually contains some albumen; the urea is diminished or has disappeared, and is replaced by leucin and tyrosin. In the normal condition of the liver it is now regarded as probable that the urea which is eliminated by the kidneys is produced in the former organs by the metamorphosis of the albuminoids. The blood contains considerable urea, and much leucin in acute atrophy of the liver. The spleen is usually, but not invariably, increased in size. The muscular tissue of the heart undergoes more or less fatty change, but this alteration is common to many acute diseases. Spots of ecchymosis form in the peritoneum, the gastro-intestinal mucous membrane, in the skin, etc., and indicate the destructive changes which have occurred in the blood.

Symptoms.—This formidable malady begins insidiously—as a simple catarrh of the stomach and duodenum, with a slightly coated tongue, nausea and vomiting, headache, tenderness of the epigastrium, and a slight icterode hue of the skin which gradually deepens. There are some acceleration of the circulation and slight fever, which, however, are not constant, for the pulse may and usually does have the feebleness and slowness belonging to jaundice. The duration of these mild symptoms is by no means constant—they may occupy a week or more; and, from the appearance of decided jaundice to the onset of the serious symptoms, there may be a few hours to two weeks. Sometimes the severe symptoms come on with the jaundice and a day or two before the temperature rises. An obstinate insomnia now begins, and the headache becomes intense. This period has, by some,* been entitled the *icteric period.* According to Frerichs, these symptoms of gastro-duodenal catarrh exist in about one half of the cases, and the duration of them may be from three to five days, although in some cases they last two to three weeks. In one case † an attack of jaundice preceded, by several months, the fully developed attack.

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* Drs. Lewitski und Brodowski—Virchow's "Archiv," Band lxx, p. 421—"Ein Fall von sogennanter acuter gelber Leberatrophie."

† Ibid., Band xliii, p. 533. Waldeyer.

A rise of temperature either precedes or accompanies the serious symptoms—the *toxæmic period.* The pulse becomes very rapid, rising to 140, but suddenly again, without any apparent reason, it may be, or in consequence of hæmorrhage, falling to 70 or 80. These fluctuations, which may occur several times a day, are peculiar to the disease. When the cerebral symptoms come on, the pulse becomes uniform at 140 to 160. The temperature line is of the remittent type, with a morning remission (102° Fahr.) and an evening exacerbation (104° Fahr.). Jaundice is constantly present, and gradually deepens from its first appearance; and intermixed with it are large brownish ecchymotic patches, but these are not always present. The tongue and gums are brownish, dry, and covered with sordes and crusts, and the breadth is fetid. There are much nausea and vomiting, and severe pain is experienced in the epigastrium and through the right hypochondrium, and pressure over the hepatic region awakens severe pain. A diminution in the size of the liver can be readily made out by percussion, and at the same time and relatively an increase in the dimensions of the spleen. There is constipation in the beginning, followed by more free, tarry stools, the product of intestinal hæmorrhage. During the first vomiting, mucus and bilious matters are discharged; but, when the toxæmic symptoms come on, blackish, grumous blood, or "coffee-grounds," are ejected. There are more or less epistaxis, bleeding of the gums, as well as vomiting of blood, and ecchymoses form at various places. The urine is usually normal in quantity, acid in reaction, and has the normal specific gravity. When delirium and coma exist, the urine is either retained or passed involuntarily. Very great changes are noted in its composition : the urea is diminished in amount, the phosphate of lime disappears, and a quantity of leucin and tyrosin and extractives are substituted. It contains also bile-pigment and traces of albumen, and cast-off epithelium deeply stained with bile-pigment. There must necessarily accumulate in the blood those excrementitious matters which it is the office of the liver to separate from the blood, and this fluid is deprived of those contributions to it made by the action of the bile in the digestion of certain aliments. We can not therefore subscribe to the doctrine of Flint, who assigns to cholesterin the toxic effects, which are doubtless produced by several excrementitious matters. Instead of the "cholesteræmia" of Flint, we hold to the older term, cholæmia or acholia. These poisonous materials act on the nervous system in a manner similar to a narcotic poison, producing at first a stage of excitation, followed by depression. A hypochondriacal state, with irritability and restlessness, is the first manifestation of mental disturbance, but this is soon followed by noisy delirium. From this state to low-muttering delirium and coma the transition is quick ; or convulsions, local twitching, cramps, and general epileptiform attacks occur, soon passing into coma and insensibility. Sometimes death takes place in tetanic spasms.*

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* Jaccoud, vol. ii, p. 418.

† Dr. Joseph Coates, "The British Medical Journal," June 26, 1875.

Course, Duration, and Termination.—The behavior of acute atrophy of the liver is irregular : the prodromic period, the stage of jaundice, and the toxæmic stage, are uncertain in duration, but the last stage follows a more uniform plan. After the development of the jaundice period, from the rise of temperature and the insomnia which mark the onset of the toxæmic stage till death, the most usual period is five days. The prodromic stage may last a week or two, the jaundice stage from a day or two to two weeks, the toxæmic stage a week, but the rule is that the whole course of the malady is included within a week. The termination is in death. Some successful cases have been reported, but it is doubtful if they were genuine. It may be that many cases treated carefully at the outset have been arrested and cured, but such cases are, as far as we are informed, simply cases of jaundice from catarrh of the bile-ducts. When the hepatic cells are disintegrated, a cure can hardly be possible.

Diagnosis.—Acute atrophy is probably more frequently overlooked than recognized. It is impossible to differentiate the gastro-duodenal catarrh of this disease from the ordinary examples of the same disease. Great importance must be attached to the increased headache, rise of temperature, and obstinate wakefulness which mark the onset of the toxæmic stage. As so many of these cases occur in pregnant women, they are apt to be confounded with puerperal fever, puerperal septicæmia, etc.; but the physical signs of a rapidly diminishing liver, the nervous phenomena, the hæmorrhages, and especially the changes in the urine, will serve to distinguish between them.

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* Morand, "Gazette des Hôpitaux," 20, 21, 1873.

Treatment.—Frerichs reports a supposed case of acute atrophy, which got well under purgatives and mineral acids. This appears to be the routine treatment. If the disease had any relation to the amount or quality of the bile, the use of podophyllin, euonymin, ipecac, and other remedies of the same group, is indicated, and mineral acids should be given freely, well diluted, in small doses frequently repeated. As the disease is a diffuse parenchymatous inflammation, the best results will be obtained from the use of a large dose of quinia and morphia in the incipiency, but will be useless when the liver-cells have begun to disintegrate. The author advises the trial of very small doses of phosphorus, as early as possible, as this remedy affects the organ specifically, and an action of antagonism may be discovered between them. This remedy, as all others, will fail to do the least good, if disintegration of the cells has occurred. Alcoholic stimulants should be pushed freely, notwithstanding a condition not unlike acute atrophy has been lately observed from subacute alcoholismus.*

AMYLOID LIVER.

Definition.—By this term is meant a degeneration of the liver caused by the deposit of an albuminoid material, termed *amyloid*, because of a superficial resemblance to starch-granules. This disease is also called "waxy liver," and "lardaceous liver," in recognition of the peculiar physical condition of the organ.

Causes.—The chief cause of amyloid degeneration of any organ is prolonged suppuration, especially in connection with diseased bone, and the morbid process is then general, the liver suffering in common with other organs. A variety of explanations have been offered to account for the production and deposit of this amyloid matter. The theory of Dr. Dickinson, which assumes that this matter is a form of fibrin, altered by the loss of its alkali, which in the normal state is intimately associated with it, has been overthrown, by the recent investigations of Mr. George Budd, Jr.† In the blood, as Seegen first demonstrated, there exists a substance—*dystropodextrin*—"which agrees with lardacein in its most essential characteristic." This material, it is now supposed, becomes insoluble and is precipitated in the textures, under those conditions with which we are now familiar as causative of the morbid state. The suppuration of tubercular cavities, of scrofulous abscesses, of intestinal and leg ulcers, etc., may also, although less frequently, be a cause of this degeneration. Next to suppuration, the most influential factor is chronic syphilitic infection, and then chronic malarial poisoning. The abuse of mercury is an alleged cause which Frerichs disposes of satisfactorily. This morbid state occurs more frequently in men than in women, and attacks by preference the most active period of life—from twenty to forty years of age.

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* M. H. Rendu, "Note sur deux cas d'alcoölisme subaigu ayant donné lieu à des accidents comparables à ceux de l'ictère grave." "La France Médicale," September 17, 1879.

† London "Lancet," February and March, 1880.

Pathological Anatomy.—The liver presents a very characteristic appearance: it is uniformly enlarged without alteration of the form and relation of its parts, and sometimes its dimensions are enormous. It presents to the naked eye a pale grayish, glistening, opaline, translucent appearance, and to the touch a doughy consistence. On section the surface is homogeneous, and resists the knife almost like cartilage, and is anæmic and whitish; and when the disease is far advanced no trace remains of the proper structure of the organ.[*] There may be parts only, or the whole organ, affected by the change. The deposits may be in patches, small or large, and restricted to parts of the organ, or be uniformly distributed through it, and may be so limited in amount as not to increase its size (Frerichs).[†] Cirrhotic or fatty degeneration may coexist with the lardaceous, when, of course, the appearances will correspond. The reaction with iodine and sulphuric acid affords a striking test of the amyloid deposits. The parts to be examined must be carefully cleansed, and a solution of iodine with iodide of potassium in water, or diluted tincture of iodine, brushed over, when they assume a mahogany color, quite different from the yellow color of the healthy tissue. This reaction may be sufficiently characteristic of itself, but, if to the iodized surface is now added some diluted sulphuric acid, the affected parts, after some minutes or hours, take on a violet tint, more rarely bluish. The violet may be very deep, almost black. Orth[‡] advises that a large and thin section be laid in a saucer of water containing some iodine, and, when the changes are complete, placed on a white plate. The reaction will be very distinct. Microscopically, the structural alterations affect first the arterioles and capillaries; their diameter is increased, the lumen narrowed, even closed; the intima, the endothelium, and the muscular coat, more rarely the adventitia, are invaded by the deposits. The cells become cloudy, granular, then clear, bright, and homogeneous, and the nuclei disappear. When the process is completed, the cell is transparent, glistening, and brittle, easily breaking up into small fragments.[§] The amyloid change is not confined to the liver, but involves the spleen, the kidneys, the lymphatic glands, the intestinal mucous membrane, and other organs. Those portions of the liver remaining unaffected by this morbid deposit are in a state of congestion, and are softer; or parts of the organ are attacked with fatty or cirrhotic degeneration, or syphilitic gummata may be mixed up with the amyloid deposits.

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[*] Wagner, "Manual of General Pathology," p. 322. New York: William Wood & Company. 1876.

[†] *Op. cit.*

[‡] Orth, "Diagnosis in Pathological Anatomy," p. 321. Riverside Press. 1878.

[§] Förster, *op. cit.*, p. 272.

Symptoms.—There are probably no exceptions to the statement that amyloid degeneration occurs in subjects already in a cachetic state by the existence of one or more of the causes already mentioned. The symptomatology is necessarily that of the malady with which this degeneration is associated, up to the time of the development of those signs by which the disease of the liver is recognized. The liver is usually enlarged, and often considerably so, extending several fingerbreadths below the margin of the false ribs. The organ is smooth, firm to the touch, almost of stony hardness, it may be; its borders well defined, free from pain or tenderness, unless there is present local peritonitis. This increase of size has gone on without any local uneasiness to call attention to the organ. The spleen is also enlarged, and is firm in texture, as a rule, but the waxy degeneration does not always affect it when enlarged in the course of amyloid liver. Jaundice is exceptional, unless the common duct or the hepatic duct is obstructed by enlarged lymphatics. As the amyloid change first affects the branches of the hepatic artery, the portal is not interfered with until later. Ascites exists in about one fourth of the cases, and is often preceded by œdema of the lower extremities, the result of a general hydræmia. The appetite is usually poor, but in exceptional cases is voracious. Food in the solid form excites uneasiness soon after it is swallowed, and is rejected by vomiting, or passes unchanged in the fæces, unless it is very bland and capable of entire solution in the stomach. The fatty, starchy, and saccharine articles of the diet undergo decomposition in the intestine, and a great deal of gas—the foul compounds of hydrogen with sulphur and phosphorus—is the result. The amount of bile passing to the intestine lessens with the increase of the deposit in the hepatic cells, and ultimately the secretion is arrested, and the office of the bile in preventing putrefaction and in emulsionizing the fats terminates. The obstruction to the portal circulation maintains a constant hyperæmia of the gastro-intestinal mucous membrane. As a result of these causes, the stomach and intestines become irritable, and frequent liquid stools, now pale from the absence of bile, now dark from the presence of blood, are passed. Amyloid degeneration also invades the arterioles of the mucous membrane and the substance of the villi, and destructive ulcers are formed in consequence (Frerichs). The urine is pale, abundant, of low specific gravity, and contains waxy casts and a trace of albumen. It is not surprising, in view of the structural alterations and impairment of functions, that the subjects of amyloid degeneration present a peculiar, anæmic, and pallid appearance, are breathless on the least exertion, and emaciate rapidly.

Course, Duration, and Termination.—As amyloid degeneration is preceded by suppuration, or some chronic wasting disease, the moment this change begins escapes recognition. Indeed, the peculiar deposits have been quite extensively distributed before any characteristic symptoms appear. When the process once begins it extends at a pretty uniform rate, and death takes place by exhaustion and general dropsy, or the end is reached by an intercurrent malady, as pneumonia, pleurisy, etc. Its course is essentially chronic ; its duration months or a year or more ; its termination fatal. Notwithstanding the unfavorable prognosis, the disease is not always fatal, and cures have been reported, especially of those cases having a syphilitic history.

Diagnosis.—The enlargement of the liver due to amyloid deposit is to be differentiated from fatty liver, hydatid disease, cancer, etc. From fatty liver it is distinguished by the greater firmness of texture, the well-defined margin, and especially by the accompanying disorders of the spleen, kidneys, and intestinal canal. From hydatid disease it is separated by the same signs, and by the characteristics of the hydatid tumor, which enlarges painlessly, is elastic, and furnishes on palpation the "purring tremor." The changes in the liver produced by cancer are secondary to the original deposit, which is most frequently in the stomach, and the enlargement of the organ is hard, nodular and irregular. The urinary secretion is not affected in cancer, but jaundice is often present.

Prognosis.—Few if any cases of true amyloid disease recover, and indeed recovery can hardly be possible when the hepatic cells are entirely filled with such a material. Cases presenting the signs of amyloid degeneration, but not far advanced, have recovered. Although the prognosis is grave, it is not necessarily fatal.

Treatment.—Prophylaxis necessarily occupies an important position in the therapeutical management of this disease. As so many—much the largest number—owe their origin to suppuration of bone and to syphilitic infection, it is highly necessary to stop the influence of these morbid processes at an early period in all cases. If there be any reason to suspect constitutional syphilis, appropriate treatment should be at once instituted, and the most efficient remedy under these circumstances is a compound of iodine : the compound solution of iodine—ten drops in water, three or four times a day, may be given ; or, if there be much anæmia, the sirup of the iodide of iron, and especially the sirup of the iodides of iron and manganese. The author has had the best results from the persistent use of the iodide of ammonium in small doses frequently repeated—five grains every four hours, and well diluted with water. Budd urges the employment of the muriate of ammonia (ammonium chloride), but the iodide, the author believes, is much more efficient. Mercurials are injurious. The diet should consist of those alimentary principles which undergo digestion and absorption in the stomach—as milk, animal broths, eggs, fish, etc.; and starches—as bread, potato and rice—sugar in any form, and fats, ought to be avoided, because they require the action of the intestinal juices. The food-supplies should be small in quantity, and given frequently, because of the intolerance of the gastro-intestinal mucous membrane. Inunction of fat, especially of cod-liver oil, is a highly useful addition to means for promoting the nutrition.

CARCINOMA OF THE LIVER.

Etiology.—Nothing is definitely known as to the origin of cancer, in any situation, but there are certain facts connected with its development which it is important to recognize. It is a disease of advanced life, and is more apt to appear from forty to sixty than at any other vigintennary. But cancer of the liver appears in early life relatively more frequently than cancer of the stomach. It occurs with about equal frequency in the two sexes. Heredity, although the fact can not be expressed in figures, is doubtless the most influential factor in its genesis.

Pathological Anatomy.—The ordinary form of cancer is found in the liver, the variety being determined by the relative proportion of the fibrous stroma, the cells, and the juice; it is most frequently medullary or encephaloid. When infiltrated with pigment it becomes *melanoid*, and, when vessels predominate, *telangiectatic* cancer, but these are accidental differences. The cancer formation may be in nodules or isolated masses, or diffused through the hepatic parenchyma. The size of the nodules varies from the dimensions of a pea to those of a child's head (Förster), and they are in numbers inversely as their size. There may be one or two of large size, or a great many of small size, distributed through the substance of the organ. Those on the surface are rounded, with a central umbilication, produced by a fatty metamorphosis of the center of the mass and contraction of the peripheral portion. The peritoneum is adherent usually, and is cloudy, thickened, and covered with a membranous exudation, or it may remain normal. The consistence of the masses varies with the form of the cancer—it is soft, brain-like, or almost creamy, or it is hard and cartilaginous. The explanation of the origin of the growth differs, but it may be stated that the cancer develops from the interlobular connective tissue. The branches of the hepatic artery are intimately concerned in the morbid process; they increase in size, and permeate the new formation, while the branches of the portal vein shrink. With the development of the cancer-cells (by division and endogenous formation of the connective-tissue corpuscles—Wagner*) the proper hepatic cells disappear. The new vessels developed from the branches of the hepatic artery have very delicate walls, and are liable to rupture, infiltrating the cancer-masses with hæmorrhagic extravasation. When the periphery of the organ is reached by the new formation, hæmorrhage may take place into the peritoneum, and sudden death ensue from this cause. The branches of the portal vein are compressed, or they may be filled with cancer-cells. The lymph vessels and glands may also become filled and infiltrated. The bile-ducts are compressed and disappear, except the larger ducts, which become dilated into pouches with retained bile, or pass unchanged through the cancer-masses. The growth of cancer is not continuous and uniform, but paroxysmal, as it were—now rapid, now slower; and when the formations have existed for some time they undergo a fatty metamorphosis. It is this change in the interior of the nodules which leads ultimately to the umbilications already mentioned. The hepatic parenchyma not invaded by the cancerous new formation remains unchanged, or is more or less hyperæmic, or undergoes atrophy. The size of the whole organ is usually increased, and sometimes it attains extraordinary dimensions, weighing ten, fifteen, or twenty pounds (Frerichs). Cancer of the liver is rarely primary, but is secondary to a deposit elsewhere, most frequently in the stomach. Of ninety-one cases collected by Frerichs, forty-six were secondary to cancer in organs having a vascular communication with the liver, and cancer was primary to the liver in scarcely one fourth of the cases. The author has met with one case of primary cancer of the gall-bladder, the morbid process *apparently* beginning in the exudation of a local peritonitis caused by the passage of hepatic calculi.

Symptoms.—Cases of cancer of the liver are occasionally encountered in which no characteristic symptoms existed; the patient has ill-defined uneasiness in the right hypochondrium, disorders of digestion, and low spirits; he emaciates progressively, is cachectic, and ultimately dies. Again, cancer of the liver has a clinical history which is merely the conclusion of a series of symptoms referable to cancer in another organ, notably the stomach. The defined symptoms of hepatic cancer are apt to be obscured by some leading condition associated with it, as ascites. Those attacked with cancer are advanced in life as a rule. Before any symptoms of disturbance in the hepatic functions manifest themselves, there are present disorders of digestion, flatulence, and constipation. Then feelings of uneasiness, of weight, of tension, and of pain in the right hypochondrium are experienced. On palpation, soreness is developed by pressure, and the liver is felt stretching beyond the margin of the ribs; it is indurated, irregular in outline, and nodulated. In the further progress of the case, the liver extends downward still more, and nodules can be easily made out; the area of hepatic dullness is increased in all directions, but chiefly downward, and there may be a good deal of spontaneous pain and exquisite tenderness on pressure by reason of a local peritonitis.

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* "General Pathology." Translated by Drs. Van Duyn and Seguin. New York, 1876, p. 503.

Jaundice is not present in the majority of cases, and exists only when the lymphatic glands in the fissure or the cancer nodules are enlarged sufficiently to compress the hepatic or common duct. Ascites is present in about one half of the cases, and is produced more frequently by peritonitis than by compression of the portal, but this vessel is obstructed occasionally by cancer thromboses. The ascites may be so considerable as to produce great distress by embarrassment to respiration and by interference with the circulation. The ascites may be in part due to the watery condition of the blood. The fluid is a pale, straw-colored serum, or it contains flocculi of lymph and is turbid, or it is mixed with blood, the source of which has been heretofore alluded to. Gastro-intestinal catarrh is set up by the congestion of the portal system; hæmorrhoids form; hæmorrhages occur from the intestinal mucous membrane, and an obstinate watery diarrhœa succeeds to the constipation which was an early symptom. All of these causes combine to produce a cachectic state. The complexion gradually assumes the characteristic earthy or fawn color, emaciation is extreme, the feebleness is excessive, the hands and feet are cold, the skin is dry and harsh, and the expression is dejected and worn.

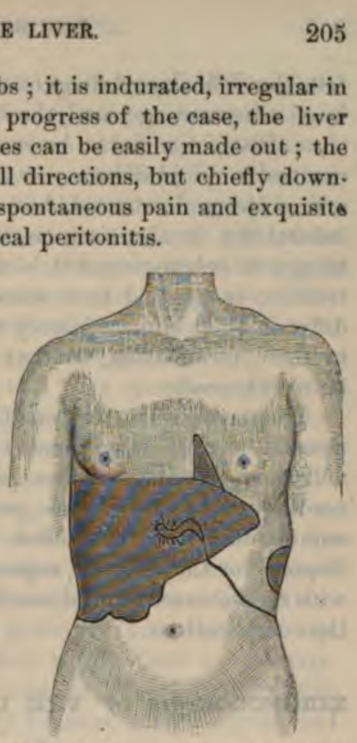
FIG. 14.—Area of Dullness in Cancer of the Liver.

Course, Duration, and Termination.—The course of cancer of the liver and its duration are much influenced by its form—the medullary proceeding to a fatal termination more rapidly than scirrhus. As already stated, the progress is not uniform, the growth at times being suspended and then again quickening into renewed activity. Cases terminating in eight weeks have been reported, and others continue with varying fortunes for months and years. There is but one mode of termination, that in death.

Diagnosis.—It may not be possible to diagnosticate cancer in those cases without any local symptoms, or in the incipiency of any case. When, however, the enlarged and nodulated liver can be felt, the difficulty of diagnosis is much less, especially if the patient is of advanced age, and the cachexia, the ascites, etc., are also present. Distinction is to be made between cancer, abscess, echinococcus, and amyloid disease ; in all these the liver is enlarged (as a rule) and projects downward, but, in cancer, the organ is nodulated and indurated ; in abscess it is smooth and softer, and may be fluctuating ; in echinococcus it is smooth, elastic, and having the purring tremor ; in amyloid it is smooth and uniform, but indurated. They differ in their clinical history and in their cause, in their duration and in their termination, so that a diagnosis can, in well-marked cases, be readily made.

Treatment.—The treatment must necessarily be palliative and symptomatic, as there is no remedy for cancer in any situation. Anodynes will be required to relieve pain. Careful regulation of the diet, according to the conditions present, and the timely administration of stimulants will be demanded. Ascites will require the treatment indicated for that disease, especially the tapping—for the interference with repose caused by a distended abdomen is one of the most distressing complications.

ECHINOCOCCUS OF THE LIVER (HYDATID DISEASE OF THE LIVER).

Definition.—By the terms echinococcus of the liver, hydatid disease, cystic degeneration, multilocular cyst, etc., is meant the penetration into the liver of the scolex of the sexually immature tænia echinococcus. The embryos, gaining access to the intestines of man, migrate, and, doubtless chiefly by the portal vein and bile-ducts, reach the liver in which the cyst or cysts develop, sometimes attaining immense size.

Causes.—As the echinococcus is the tænia of the dog, only those who live in a humble way, with their animals about them, suffer from these migratory parasites. As the ova are discharged with the excrement of the dog, it is obvious that they can gain admission to the human stomach only through the most filthy practices, or by carelessness in the obtaining and storing of drinking-water and food. In Iceland, more than in any other part of the world, do the people suffer from cystic disease—as large a proportion as one sixth of the population being infected. This preponderance of the disease is due to the number of dogs and to the promiscuous way in which the members of a family and their dogs live together in their wretched hovels. The disease occurs at the middle period of life chiefly, and rarely in the young. In the only case of echinococcus of the liver met with by the author, the patient, a male, was forty-two years of age.

Pathological Anatomy.—When the echinococcus (or two or more) lodges in the liver it is presently enveloped in a tough, fibrous, yellowish-white membrane, constructed out of the adjacent connective tissue, and closely adherent. Within this adventitious membrane is contained the embryo, inclosed in a clear, translucent sac made up of numerous concentric layers. This sac of the embryo is the mother-sac, and in the interior of it a number of so-called daughter-vesicles, and still other, granddaughter-vesicles, are developed, and ultimately the mother-sac, with its investing membrane, attains to extraordinary dimensions. The daughter-vesicles vary in number from a few up to many thousands, and in size from that of a pea to that of a goose-egg. The fluid of the sac is clear, opalescent, weakly alkaline, and of a specific gravity of 1·008 to 1·013 ; it contains no traces of albumen, but a large proportion of sodium chloride and some crystals of cholesterine and hæmatoidine.* The inner membrane of the daughter-vesicles is lined with a germinating layer, from which the embryos spring ; and scolices, attached as well as free, can be observed within the sacs. These scolices are the immature tæniæ, and can be recognized with a low power—sixty diameters—as possessed of a head, four suckers, and a row of hooklets. When detached, these scolices have the power of active motion, and can withdraw their probosces and hooklets within their own cavity. There are hydatids without daughter-vesicles, and others entirely without a scolex, which were denominated by Laennec acephalocysts, and by Küchenmeister,† sterile echinococci. There are great variations in the size, number, and position of the cysts. They are found in all the lobes, but most frequently in the right, buried in the substance or projecting from the surface of the organ. Usually but one cyst exists, but there may be several—as many as five or six. It follows that the size, shape, and appearance of the liver will vary with the number, position, and growth of the cysts. It may attain a sufficient size to distend the abdominal cavity, or at least make a great protrusion in the right side. With the growth of the cyst, the hepatic tissue is correspondingly atrophied, by being encroached upon, while the rest of the organ remains intact, or undergoes hypertrophy, or is hyperæmic. As a rule, the cysts do not obstruct the large blood-vessels and bile-ducts ; hence the infrequency of ascites and jaundice ; yet both may be encroached upon—even obliterated. It sometimes happens that communication is established between bile-ducts and the cyst, by the breaking through of the duct in the course of development of the cyst, and, bile entering, the growth of the echinococcus is arrested. The cysts sometimes penetrate the common duct, also the gall-bladder, and rarely the portal vein. They may be discharged through the ducts and a cure be thus effected, but, if they enter the veins, thrombi form, with the usual disastrous results. Echinococci-cysts may undergo calcification. The adventitious envelope becomes thicker and tougher, and calcareous salts are deposited; expansion and growth are prevented; the parasites die, and are found flattened and contracted. In other cases there is developed in the interior of the capsules a dense, honey-like or puriform fluid, which had previously been clear and then milky, and remains of the scolices, especially the hooklets, are found floating in, or mixed with, the contained fluid. Crystals of hæmatoidine and bile also are found mixed with the contents of wasting cysts.

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* Davaine, "Traité des Entozoaires." Paris, 1872, p. 379.

† "Animal and Vegetable Parasites," *op. cit.*

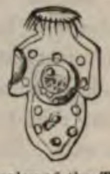

FIG. 15. Isolated Scolex of the *Tænia echinococcus*, from the Pig.

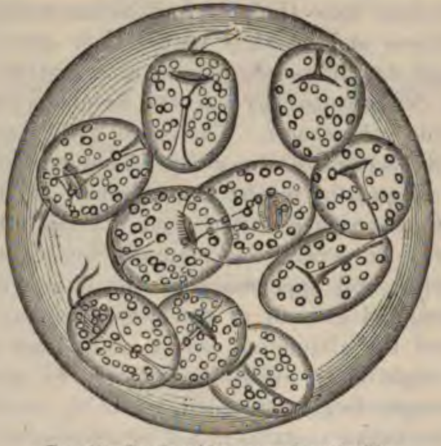

FIG. 16.—*Tænia echinococcus*, from the Pig.

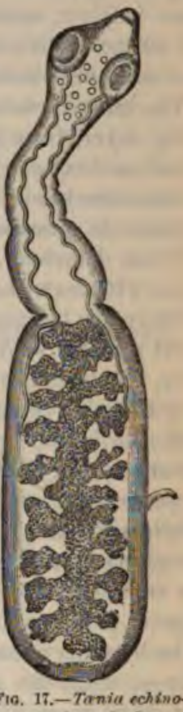

FIG. 17.—*Tænia echinococcus*, from the Dog.

A great many cysts are destroyed and cease to grow, as has been described, but many continue to enlarge, pushing up the diaphragm and displacing the heart, and reaching sometimes as high as the second rib (Frerichs). Others, growing downward from the under surface of the liver, push aside the stomach, and force the abdominal organs into the pelvis, or, but rarely, compress the ascending vena cava, causing œdema, varicose veins, etc. A cyst may rupture into the cavity of the chest—into the pleural or pericardial sac, causing fatal inflammation, or excavate a cavity in the right lung, and shreds and parts of the vesicles be discharged through the bronchi by expectoration. A cyst may also rupture into the peritoneum, producing fatal peritonitis, or into the intestines, and be slowly discharged by stool. Rupture within the abdomen is usually due to a blow or other injury, but is sometimes spontaneous. The *echinococcus multilocularis*, which was formerly mistaken for colloid cancer, but has since been accurately described by Virchow, differs from the ordinary form, in that it is a very firm, hard tumor, consisting of dense fibrous tissue, containing cavities filled with a gelatinous material. On account of its tendency to ulcerative degeneration, Virchow called it the "ulcerative multilocular echinococcus-tumor." Friedreich * holds that the development of this form takes place in the gall-ducts and blood-vessels.

Symptoms.—A cystic tumor of small size, deeply placed, and not so situated as to interfere with other parts, may not cause any symptoms, and therefore remain undetected. But a cyst of considerable size, projecting from the liver, or which has increased the size of the organ, and especially if it has encroached upon neighboring parts, will cause sufficient disturbance of function to lead to its early recognition. If a cystic tumor increases to any considerable extent the volume of the liver, there will be a feeling of weight, heaviness, and dragging in the right hypochondrium, and some disorders of digestion; if it happen to be near the hilus of the organ, the portal vein and the common or the hepatic duct may be pressed upon, causing ascites and jaundice; if near or at the upper convex surface of the right lobe, the diaphragm will be pushed up, and a dry cough and dyspnœa will be the result. The degree of enlargement is necessarily various. The tumor may fill in the whole space from the inferior border of the second rib to the pelvis, displacing the thoacic and abdominal organs, and forcing out the intercostal spaces. The tumor may take various forms: the liver may be uniformly enlarged; there may be a growth projecting from the borders of the organ, and having a globular or hemispherical form similar to that of the gall-bladder; or, one lobe may be the seat of the growth, the other remaining intact.

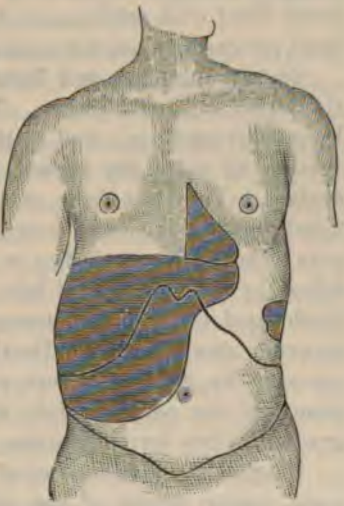

FIG. 18.—Liver enlarged by Hydatid Cysts.

On palpation, an hydatid tumor is elastic, resisting but soft, fluctuating, and, in somewhat more than half the cases, presenting the peculiar fluctuation known as "purring tremor," or "hydatid purring"—a sensation appreciated by the sense of touch as the trembling of a bowl of jelly appears to the eye. The tumors are not painful, and it is exceptional for any tenderness to be felt on pressure. Jaundice or ascites occurs only in the rather rare event of a tumor near the hilus, or so situated as to compress the vein and duct. Dyspnœa and cough occur when the cyst develops into the thorax; irregular action of the heart, when this organ is pushed from its position; constipation and vomiting, when the intestines and stomach are encroached upon; swollen and œdematous feet and ankles and enlarged veins, when the cava is compressed. All of these symptoms arise, when the form and direction of the cyst develop them, without any constitutional disturbance, and if such disturbance occur it is due merely to the interference of the growth with important functions. If the echinococcus burst, new symptoms arise. If the stomach is entered, there will be some local pain, and the parasites will be rejected by vomiting, often in immense numbers; if the intestine is perforated, the parasites are discharged by stool, and recovery may ensue in either case. If the vena cava is entered, sudden death with the symptoms of asphyxia takes place. If the pleural cavity receive the echinococci, pleuritis is excited, and the cysts, with the products of inflammation, may be subsequently discharged through the lung by a bronchus. If the pericardium is suddenly filled with echinococci, the action of the heart is disturbed, and fatal pericarditis quickly excited.

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* Virchow's "Archiv," vol. xxxiii, p. 16, "Ueber multilokulären Leber-echinokokkus."

Course, Duration, and Termination.—The hydatid disease is essentially chronic in its course. The development of the cyst is affected by its surroundings; and in the interior of organs, subjected to pressure on all sides, the growth is slower than if it is deposited on the surface. They last from one or two years up to thirty, but the most usual duration is two to four years. They may undergo a spontaneous cure: the echinococci die, or on the opening of bile-ducts they are killed by the entrance of bile, and subsequently shrivel up; they are discharged through the stomach and intestine, or by the bronchi, and recovery slowly ensues. Death is not unfrequently produced by echinococci—by gradual failure of the powers of life; suddenly, by entrance of the parasites into the vena cava or the pericardium; and gradual failure by pneumonia, or suppuration, or pyæmia.

Diagnosis.—Echinococci of the liver may be confounded with abscess, cancer, dropsy of the gall-bladder, aneurism, and hydrothorax. It differs from abscess, cancer, and hydrothorax by the absence of pain and constitutional disturbance; from abscess, by the character of the fluctuation; and from cancer, by absence of the hard, non-fluctuating nodules of the latter. From dropsy of the gall-bladder it is distinguished by the lack of a history of attacks of hepatic colic, their cessation and the enlargement of the gall-bladder coming on slowly; but the distinction is most certainly made by the use of the aspirator, since it has been shown that this organ may easily and with perfect safety be penetrated by the needle. From aneurism, echinococci are readily differentiated by the existence of a heaving, expansile pulsation in the former, without the peculiar fluctuation of the latter. There is more real difficulty in separating hydatids pushing up the diaphragm, from effusions into the pleural cavity, as the physical signs are the same. An attentive consideration of the previous history will aid materially in arriving at conclusions. The growth of echinococcus is slow and painless, and the development of the local symptoms is free from that disturbance which precedes the occurrence of an effusion in the chest. But, above all other means for coming to a correct conclusion, must be placed the use of the aspirator and the microscopic examination of the fluid.

Prognosis.—When the echinococcus is large, and its particular direction unknown, the prognosis is grave. The early use of the aspirator enters largely into the question of prognosis, for early puncture will insure the death of the parasite. When discharge takes place by the stomach and intestine, the prognosis will be favorable; and recovery may also be expected in those cases discharging by the bronchi, provided the right lung is only so far damaged as to permit the passage of the cysts. When there is a large suppurating cavity in the right lung the prognosis is unfavorable.

Treatment.—There is no medicinal treatment which can in any way affect the origin or growth of the echinococci. Fortunately, we possess simple surgical measures by which these cysts may be safely and certainly closed. These are, puncture by an aspirator needle and withdrawal of some of the fluid, and electrolysis. Whenever a cyst can be reached by the needle, it can be subjected to either of these expedients. The simple puncture and withdrawal of some of the fluid contained in the mother-vesicle should be tried first, as this has succeeded in numerous instances. This failing, the method by electrolysis should be practiced. Dr. Hilton Fagge and Mr. Durham * report eight cases in which electrolytic decomposition was employed with entire success. Two needles connected with the negative pole were inserted into the sac, and the positive pole, in the form of a large sponge-electrode, was applied on the integument in the neighborhood. Ten cells were used to furnish the current, and the needles were permitted to remain ten minutes. As, in the process of electrolytic decomposition, hydrogen and the alkalies (potassa, soda) appear at the negative pole, it is obvious that the parasites must be killed by the electrolytic action. Besides these measures, iodine has been injected into the mother-sac with success.

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* "Medico-Chirurgical Transactions," vol. cliv, "On the Electrolytic Treatment of Hydatid Tumors of the Liver, with an Addendum on Simple Acupuncture."

ANEURISM OF THE HEPATIC ARTERY.—The author can add one to the few examples of aneurism of the hepatic artery. The size of the tumor in the reported cases has varied, but the tumor can not always be felt, or rupture takes place before it has attained sufficient dimensions to be felt through the abdominal parietes. In one instance the liver was displaced by it. Usually, long before the existence of a tumor can be made out, severe pains are experienced in the right hypochondrium. The attacks of pain are at first paroxysmal, and can hardly be distinguished from hepatic colic, but in the further progress of the case there are constant pain and soreness in the right hypochondrium, and paroxysms of severe pain. The pressure of the aneurism on the hepatic plexus is the cause of the early appearance, severity, and persistence of the pain. Jaundice is usually present, due to pressure on the hepatic or common duct, and, in the case referred to by the author, ascites was the prominent symptom. The interference with the hepatic functions, the constant suffering, etc., cause rapid failure of the vital powers; the flesh wastes, the skin appears earthy or jaundiced, the digestive functions are disordered in consequence of the absence of bile, and ascites may slowly accumulate. Death takes place by rupture and escape of the blood into the peritoneal cavity. In one case (Frerichs) blood was regurgitated by the stomach, and it reached this organ by a circuitous channel; communication by a very small orifice was established between the sac of the aneurism and the gall-bladder, and a small quantity of blood continually passed from the gall-bladder to the duodenum, and thence by retching into the stomach.

THROMBOSIS OF THE PORTAL VEIN is a result of various obstructive conditions, as cirrhosis, chronic atrophy, cancer, and tumors. The symptoms due to the thrombosis are those of obstruction to the portal circulation, and occur rather abruptly in the course of the chronic malady associated with it. The pressure in the initial radicles of the portal vein is suddenly increased, and free transudation of blood occurs along the intestinal mucous membrane, hæmorrhoids form, and a watery diarrhœa takes place. The spleen enlarges, and ascites develops with great rapidity. Efforts toward a compensatory circulation are made by the communicating veins, which suddenly appear enlarged on the surface of the abdomen. The urine becomes scanty and of high specific gravity. The patient presents a very decided cachexia, the strength rapidly fails, and death occurs in a few days or weeks. The obstruction by the thrombus is not always complete, so that an imperfect circulation is maintained. In that case the symptoms will be less formidable and the progress less rapid. The only remedy which offers any prospect of relief is ammonia, which has the power to dissolve coagula. Unfortunately, the stasis in the portal system so hinders absorption that remedies do not readily enter the blood. As Halfourd, of Australia, has demonstrated the innocuousness of the intravenous injection of ammonia, this expedient should be practiced in such cases. It consists in the injection of one part of aqua ammoniæ to two parts of water into any convenient vein. If, however, there be any movement of blood in the portal, the ammonia should be administered in the form of the carbonate — five grains every three hours. The usual remedies for ascites will be necessary.

SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN, or SUPPURATIVE PYLEPHLEBITIS.—This is always a secondary disease, and has its origin in suppuration occurring at some point in the distribution of the portal vein. An inflammation occurs in the tunics of the vessel, which become soft and discolored by the presence of a fluid and fibrinous, purulent exudation, and by imbibition of the hæmatine. The intima especially is discolored, brownish, yellowish, or greenish-yellow, and is covered with layers of fibrin and pus. The changes extend to and involve the adventitia. A thrombus forms in the vessel and undergoes characteristic alterations, softens in the center, becomes yellow, the fibrin breaking up into a granular mass, and the hæmoglobulin disintegrating and gradually forming, with the rest of the thrombus, a purulent-looking fluid. Thrombi form most frequently in the hepatic branches of the portal, and emboli in some cases are deposited in other parts of the liver, and secondary pyæmic abscesses occur in various parts of the body.

Suppurative inflammation of the portal vein is associated with and is dependent upon ulcerations in various parts of the intestinal mucous membrane, or suppuration and abscesses in the mesenteric glands, or the inflammation and ulceration following impaction by gall-stones, etc. The symptoms, therefore, succeed to those of the malady which caused it. The initial symptom is pain, and it is felt in the umbilical region, in the iliac region, or in the hypochondrium, according to the branch of the portal implicated; then follows a severe rigor, which, after a period of high temperature, terminates in a profuse sweat. These paroxysms, intermittent in type, are repeated, not in a regular order, but at uncertain intervals. In the interval the temperature is rather subnormal; during the pyrexia the temperature rises to 105° or 106° Fahr., and the sweats are most exhausting. The liver enlarges and is tender, and jaundice appears. The spleen also enlarges, doubtless because of the obstruction in the portal circulation. Usually there is a profuse diarrhœa, the discharges consisting of a reddish, watery, and fetid fluid, sometimes of bilious matter. The abdomen becomes tender, and is much distended; vomiting comes on; the exhausting alvine discharges continue, and hence the powers of life rapidly decline.

The secondary deposits excite local distress, and each addition to the area of suppuration increases the hectic fever. Deposits in the brain cause delirium and stupor, but, without these, low-muttering delirium comes on, with a typhoid state, and death occurs in a gradually deepening coma. The fatal result may occur in one week, or may be postponed to six weeks—the average being about three.

The diagnosis must always be a matter of extreme difficulty, and can, indeed, be made only when the cause is clear and all the symptoms appear in their proper relation. It will be impossible in any doubtful case to differentiate between pylephlebitis and abscess of the liver.

The treatment is without utility. While this is true, it is certain, however, that much may be done to relieve pain by the hypodermatic injection of morphia. It is in a high degree probable that large doses of quinia may be very serviceable in checking suppuration, and the free use of alcohol is certainly applicable in the same direction. The combination of morphia and quinia, with the conjoined administration of alcoholic stimulants, offers the best prospect of relief.

DISEASES OF THE BILIARY PASSAGES: CATARRH OF THE BILE-DUCTS.

Definition.—By catarrh of the bile-ducts is meant an inflammation of the mucous membrane, with an increased production of mucus. Very rarely there occurs a croupous inflammation, associated with infectious maladies, as pyæmia, diphtheria, etc.

Cause.—Catarrh of the biliary passages may arise spontaneously from climatic causes or from malarial influence. It occurs, therefore, more frequently in the autumn, when cool nights succeed to warm days, and when malaria is most rife. Malaria may induce jaundice by catarrhal swelling of the bile-ducts, without any febrile disturbance.* Catarrh of the bile-ducts is usually a secondary disease, secondary to duodenal or gastro-intestinal catarrh, which extends by continuity of tissue up the bile-ducts. A variety of causes are concerned in the production of duodenal catarrh—notably, excesses in eating and drinking. Usually the attacks are excited by some article of food which especially disagrees, but a catarrhal state of a chronic kind has preceded the acute attack.

Pathological Anatomy.—More or less extensive hyperæmia is the initial lesion. The common duct is more affected than any other part of the canal-system, but the catarrhal process may extend to and involve the canaliculi. The mucosa is swollen, the more decidedly near the duodenum, and is coated with a tenacious mucus, so that the lumen is much narrowed or obstructed. The mucous secretion of the gall-bladder is increased in amount and mixed with the bile, stored up more abundantly because the obstruction at the outlet existed while the hepatic and cystic ducts were still pervious. The viscid mucus and sero-mucus poured out from the surface of the membrane contain cast-off epithelium, abundant nuclei, and white corpuscles, and the endothelium itself undergoes proliferation. The obstruction below preventing the escape of bile, and the mucus and sero-mucus accumulating by continued production, the ducts above become dilated, and the tissue of the liver presents the usual appearance of bile-staining when there is a biliary stasis. After several days the hyperæmia lessens, and a quantity of dead endothelium is cast off, still more effectually blocking the passage; but the contents of the bile-ducts gradually liquefy, and the lumen is restored to its former dimensions by the escape of these matters into the duodenum. The whole process will occupy several weeks. This fortunate solution of the catarrhal process is not always effected. The soft tissue of the liver-parenchyma is exceedingly liable to degenerative changes. Recent researches (Charcot,* Legg †) have demonstrated that mere mechanical blocking of the common duct leads in a short time to fibroid degeneration (increase of the connective tissue, interstitial hepatitis) and atrophy of the gland-cells. It has long been known that persistent attacks of catarrh, or the frequent repetition of them, will lead to changes in the parenchyma; but these late investigations, by demonstrating the readiness with which pathological alterations occur in the hepatic parenchyma, have added much to the pathogenetic importance of catarrh of the bile-ducts. Rarely, isolated portions of the liver remain obstructed, and dilated ducts, surrounded by parenchyma deeply stained with bile and much altered, exist in patches throughout the organ.

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* "Des Affections Paludéennes du Foie," par MM. A. Kelsch et P. L. Kiener, "Arch. de Physiologie normale et pathologique," 1878, p. 571, *et seq.*

Symptoms.—The signs and symptoms indicating the onset of the malady are not the same for all forms. The form due to alternations of temperature at certain seasons commences abruptly with some pain, soreness, and sense of weight in the right hypochondrium; constipation exists, the tongue is coated, and the appetite absent; and there are some feverishness and general *malaise*. There are also much depression of spirits and a feeling of illness, greater than the actual lesions warrant. In from three to five days the eyes become yellow, and icterus, or jaundice, then gradually appears over the whole body. Usually the fever disappears in two or three days, the skin becomes dry and harsh, and the surface cold. The pulse is slow, the action of the heart weak, and the strength depressed. When this form of jaundice is produced by malarial infection, the symptoms will develop more slowly, unless, indeed, the disturbance in the hepatic functions is accompanied by malarial fever—intermittent or remittent. The most usual determining cause of catarrhal jaundice is gastro-intestinal, especially duodenal, catarrh. In some subjects a chronic catarrh exists, and but little additional disturbance suffices to close the duct. In others an acute catarrh is brought on by some indigestible food or improper drink. In either case, the patient experiences a good deal of nausea, has a heavily coated tongue, headache, and a somewhat muddy complexion, and there may be more or less fever, or none at all. The jaundice does not appear at once; there must be sufficient time for the extension to the bile-ducts to take place, which will require from one to two weeks. The bile-pigment tints all the tissues of the body, the secretions, and even pathological products, as effusions into the ventricles and thoracic cavity. The urine soon assumes a brownish color, like that of port or black coffee, and is heavily loaded with urates. Some drops of the urine placed on a white porcelain surface, and a little nitric acid made to flow against it, will exhibit the following reaction at the margin where the two fluids come in contact: a greenish tint, quickly followed by blue, violet, to red. This play of colors may not be seen, but bilirubin, where touched by nitric acid, should take on a greenish hue, being converted to biliverdin. During the febrile stage, if fever has existed at all, the pulse rises; but when jaundice appears, if no fever is present, the action of the heart is slowed and the tension of the vascular system lowered. The pulsations may decline so mnch as twenty or thirty to the minute. This depression of the circulation is due to the action of the biliary salts on the heart itself, for the same effect is produced when the pneumogastric has been previously divided. No bile passing into the intestine, certain substances fail to be digested, especially the fats, and the foods present there decompose, and a great quantity of fetid gas is formed. The results, then, of the absence of bile are white, pasty, or grayish-white, or gray, slate-colored stools, having a very offensive smell, and flatulence. The presence of bile in the skin excites in most persons a great deal of unpleasant itching, which may, indeed, be troublesome enough to prevent sleep. The vision is yellow from the presence of bile-pigment in the humors of the eye. The liver increases in size, and extends a little beyond the margin of the ribs, and the gall-bladder is also sufficiently distended to be felt, in thin persons, projecting beyond the margin of the liver, or be made out by careful percussion. If the gall-bladder partakes in the inflammation, it becomes tender. Usually in from two to five days after the jaundice appears, the unpleasant symptoms subside—the fever ceases, the tongue cleans, and the appetite returns, and only the jaundice and the torpid state of the intestines remain. In a few days the stools become darker and then normal, the fetid odor disappearing at the same time. The coloration of the tissues and the pigment in the urine continue until the work of elimination is complete, and hence high-colored urine is the final symptom.

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* "Leçons sur les Maladies du Foie, des Voies Biliaires et des Reins," Paris, 1877, p. 354.

† "St. Bartholomew's Hospital Reports," vol. ix; various articles in the "British Medical Journal," etc.

Course, Duration, and Termination.—Cases pursuing the ordinary course, having the catarrhal period, the jaundice period, and the period of convalescence, last from three to six weeks, and terminate in complete recovery. Not all cases pursue this favorable course. The resolution may be postponed, and the case assume a chronic character, leading to changes in the hepatic parenchyma, consisting in increase of the connective tissue and an atrophy, largely fatty, of the hepatic cells. The existence of a chronic catarrh of the duodenum invites attacks of acute catarrh involving the ducts, the result being the same—changes in the hepatic parenchyma. Catarrh of the bile-ducts becomes much more important from this point of view.

Diagnosis.—At the beginning, catarrh of the biliary passages may be confounded with the initial symptoms of acute yellow atrophy, but the sex and the condition of pregnancy are so influential in causing the latter that we have in these etiological factors means of differentiating in two thirds of the cases. The subsequent behavior of the two maladies differs so widely as to eliminate all doubt. When the jaundice appears there is a possibility of confounding it with the jaundice which sometimes comes on in the course of cirrhosis and cancer, but an attentive examination of the course and history of each will prevent error.

Treatment.—This is one of the very few conditions in which mercurials may be prescribed in hepatic diseases, not with the view to increase the outflow of bile, but to allay irritation of the mucous membrane. From $\frac{1}{12}$ to $\frac{1}{6}$ grain of calomel, rubbed up with a little sugar, may be administered every four hours for a few days. Simultaneously, whether malaria is or is not an element in the case, two antipyretic doses of quinia (10—15 grains) should be given daily until jaundice appears, and for a few days subsequently to its full development. To maintain free action of the kidneys by salines is highly useful by favoring elimination. The ordinary effervescing powder, or the aperient effervescing powder, if constipation is decided, is well adapted to accomplish the object. The Saratoga waters, or Vichy, or Kissengen, or Carlsbad, may be drunk freely to accomplish the same purpose. In the chronic cases, with persistent plugging of the bile-ducts, which means also persistent jaundice, the most effective remedy is sodium phosphate in ʒ j doses *ter in die*, and kept up until the jaundice declines. Sulphate of sodium, also, is strongly recommended. But the sodium phosphate is the most appropriate and effective remedy in those cases of chronic gastro-duodenal catarrh with occasional attacks of catarrhal jaundice. Recent experimental (Rutherford) and clinical experience has shown the value of euonymin and iridin as cholagogues. Two grains of the former and four of the latter, given at night, and followed by a saline, afford excellent results. The mineral acids were formerly held in great esteem in the treatment of these hepatic affections, but it is now known that alkalies are more serviceable. The local application of the acid-bath to the right hypochondrium is an excellent counter-irritant, but the difficulty experienced in preventing injury to the clothing is a strong objection to its use. Careful regulation of the diet is most necessary. Solid food should be withdrawn for the time being, and all fatty, saccharine, and starchy substances also, for these require the action of the bile either for their solution and absorption, or to prevent their decomposition. The most suitable aliments are skimmed milk and beef-juice. The former should be given freely every three hours, and, if the stomach is irritable, a little lime-water should be added. The utility of the milk is twofold—as an aliment and as a diuretic. Bitartrate-of-potassium lemonade is an excellent diuretic in these cases to remove the last staining of the bile. As the catarrhal inflammation subsides, the diet may be increased, but it should consist of milk, eggs, fresh meat, fresh fish, and the succulent vegetables.

OCCLUSION OF THE BILIARY PASSAGES.

Causes.—The pressure of tumors, as cancer of the pancreas, aneurism of the hepatic artery, etc., is an exterior cause ; the impaction of a calculus, adhesion of opposed surfaces in exudative inflammation, etc., are internal causes of occlusion of the bile-ducts.

Results of Occlusion.—The mucus formed all along the canals contributes somewhat to the accumulation of fluids when the outlet is closed, but the chief constituent is bile. The neck of the gall-bladder is not unfrequently closed by an impacted calculus, the sac becoming enormously distended with a transparent, faintly greenish fluid, resulting from the transformation of the mucus and of the bile stored up before occlusion. The author has seen one example of occluded orifice of the cystic duct, in which the contents of the gall-bladder consisted of forty-four biliary calculi without any fluid. As the gall-bladder is an organ of convenience and not of necessity, its closure does not disturb the hepatic functions. It forms sometimes—for the secretion of mucus continues—a tumor of considerable size, and pyriform shape, which may be felt projecting from under the liver. Occlusion of the common duct (ductus choledochus) or of the hepatic duct leads to dilatation of the biliary passages and to changes in the structure of the liver. The whole organ is at first enlarged, but it subsequently undergoes atrophy by the pressure, and death ultimately ensues from the blood-poisoning.

BILIARY CALCULI (CHOLELITHIASIS—GALL-STONES).

Causes.—In the normal state the bile does not contain any formed constituents. The formation of calculi or concretions is determined by the precipitation of a crystallizable substance from the bile—cholesterine—which is held in solution by glycocholate of soda. The mucus formed in catarrh of the biliary passages effects a decomposition of this compound. It is probable that this result is promoted by changes in the composition of the bile, and that the cholesterine may be in excess, and hence held feebly in its combination. Calculi form more frequently after than before the middle period of life, for then cholesterine becomes more abundant; and they are encountered in the obese, in hearty feeders by preference, and in the sedentary. Females are more liable than males, especially fat women who eat rich food and take no exercise.

Pathological Anatomy.—Cholesterine is the principal ingredient of biliary calculi, and exists in the crystalline form chiefly. The actual proportion of this constituent to the others is from seventy to eighty per cent. More or less bile-pigment enters into their formation; also the carbonate of lime and earthy phosphates and carbonates; and a particle of mucus or some foreign body is the nucleus about which the other materials crystallize or aggregate. Occasionally there is a single concretion of large size, which fills the gall-bladder, but usually they are very numerous—sometimes amounting to five or six hundred. When there is a single gall-stone it is ovoid or globular, to adapt it to the shape of the sac, but, when there are several, they assume the octahedral shape, with smooth facets. They do not always assume regular shapes: some are covered with warty masses; others are leaf-shaped, etc. In color they are brownish or yellowish-brown, but in exceptional instances are found in all colors from white to black. They are very light, the specific gravity varying from 1·500 to 1·800.* Gall-stones usually contain a nucleus, composed for the most part of mucus, and cholesterine and bile-pigments are deposited in alternating, concentric layers around it. The nucleus is not always in the center, and there may be several nuclei, and hence the arrangement of the layers is irregular, and there may be deposits of earthy matter and pigment, without cholesterine, etc. Gall-stones may be found in any part of the biliary passages. They are rare in the interior of the liver, and they are not often found in the hepatic duct, because of the increasing caliber below, but are found usually and in the largest numbers in the gall-bladder. By pressure the walls are irritated and a catarrh is set up, and also ulcerations of the mucous membrane of considerable depth and extent are induced. The walls of the gall-bladder, excited to frequent expulsive efforts, undergo hypertrophy, and the mucous membrane becomes reticulated. Inflammation of the peritoneal investment is excited, and the remains of exudations and adhesions are usually found. Not unfrequently the mouth of the gall-bladder is occluded by an impacted calculus, or permanently closed by inflammatory adhesions. The gall-stones may be forced down, producing pains in the passage through the cystic duct, or, the mouth of the gall-bladder being closed, they remain and produce no further mischief. Gall-stones may become impacted in the cystic, hepatic, or common duct; inflammation and ulceration, with perforation, result.

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* Thudichum on "Gall-stones," p. 10.

Symptoms.—When gall-stones are free in the biliary passages without obstructing them, they give rise to some pain in the right hypochondrium of an intermittent character, and pains radiating thence to the shoulder, umbilicus, lumbar region, etc. There is present usually nausea, even vomiting, and there may be chills, followed by fever and sweats. These symptoms are due to the irritation of the ducts, without their occlusion. If concretions are impacted in the hepatic duct, there are pains, jaundice, and enlargement of the liver. When calculi escape from the gall-bladder into the cystic duct, if of sufficient size to irritate the mucous membrane and excite spasm, the phenomena of *hepatic colic* ensue. Sometimes, after a fit of anger, or the receipt of evil tidings, but most frequently in about three hours after a meal, a pain of exceeding violence is suddenly felt at the margin of the liver and in the right portion of the epigastric region. The pain has a boring, burning, lancinating character, and radiates through the abdomen and chest and into the shoulders and back, but the situation of the greatest anguish is in the region of the gall-bladder. The pain is so atrocious that the patient writhes with the agony, rushes up and down the room, or tosses from side to side if in bed. The surface is cold and covered with a cold sweat, and often a severe rigor occurs simultaneously. There may be clonic spasms affecting the right side, or an epileptiform seizure, with loss of consciousness, may occur. Intense nausea accompanies the pain. At first the food is thrown up, but presently, after repeated retching, only some mucus, acid and watery; but the vomiting affords no relief. The action of the heart is feeble, and the circulation is correspondingly depressed. The severity of the seizure is influenced by a variety of circumstances—by the size and roughness of the concretion, by the length of canal to be traversed, and by the condition of the nervous system. The duration of the seizure varies from a few hours to several days, and the first attack is apt to be more severe than any succeeding one. When the attack continues for several days, the pain does not always persist even for hours, for there are remissions in which only an acute soreness remains, and the exacerbations behave as regular attacks. It is highly probable that in these cases several concretions are passed in succession. Again, when the calculus passes from the cystic to the common duct, there is a feeling of relief, but a new paroxysm occurs when the calculus becomes engaged in the duodenal orifice of the ductus choledochus. Inflammation in the peritoneum may be excited about the site of impaction, and involve the neighboring structures, or the duct may become gangrenous. The calculus, by preventing the outflow of bile in the hepatic or common duct, causes jaundice, which is not a usual symptom in impaction of the cystic duct; although it may be present, the surrounding swelling being sufficient to prevent the flow of bile through the common duct, or it is probable that jaundice may be due to the disturbance in the hepatic plexus of nerves. The pain suddenly ceases sometimes by the dropping of the concretion into the duodenum. Jaundice usually succeeds to the pain, and is not often seen during the time of greatest suffering. Sometimes a calculus will remain impacted in the common duct for weeks or even months; jaundice persists, the bile accumulates, the ducts dilate, until suddenly the impaction is overcome, and violent bilious vomiting and diarrhœa announce the delivery. When the concretion remains permanently impacted, the liver undergoes the changes already noted; the connective tissue multiplies, the gland-cells waste and undergo fatty metamorphosis, and the organ shrinks in size (Charcot). Careful search should always be made in the evacuations for the calculus. The fæces should be thoroughly mixed with water, the solid particles allowed to subside and the fluid portion poured off, and this operation must be repeated until the last solid parts are reached. Sometimes—most frequently, probably—there is but one calculus, but there may be a hundred. A marvelous change takes place in the patient as soon as the calculus reaches the intestine. The pain ceases, as well as the nausea and vomiting, the bowels act spontaneously, the appetite returns, the jaundice soon disappears, and the state of health is fully restored.

Course, Duration, and Termination.—From the initial pain to the termination of all symptoms may not be longer than two days, or, if jaundice is present, five days. If a number of calculi pass, the duration of a case is indefinitely prolonged. The severe cases of this kind last several weeks. The usual termination is in health, but death from ulcerative perforation and subsequent peritonitis is not uncommon. Now and then a calculus ulcerates through the duct; in the peritonitis which follows, adhesions are formed, limiting the mischief to the immediate neighborhood; a purulent depot is thus created, and gradually a fistulous communication externally is established, and the calculus is discharged with the pus. Sometimes such a purulent depot opens communication with the intestine, stomach, or bladder. The last-named terminates fatally; the discharge by the stomach, intestine, and externally is often successful. After the calculus reaches the intestinal canal, it may serve as a source of new mischief by forming the nucleus of an impaction of the bowel.

Diagnosis.—The only maladies with which hepatic colic may be confounded are hepatalgia, gastralgia, and enteralgia. The locality of the pain, the absence of local soreness, the absence of jaundice, the absence of calculi in the stools, separate these neuralgic affections from hepatic colic.

Prognosis.—A favorable opinion may be expressed in most cases, but the prognosis must be guarded when the pain does not yield, and when the vital powers begin to flag, especially if local tenderness and fever indicate peritonitis.

Treatment.—The severe pain demands immediate attention. There are two methods of relieving it: by the inhalation of ether, and by the hypodermatic injection of morphine. The action of the former is temporary, and, of course, the relief is confined to the period of unconsciousness. This may be sufficient, but usually prolonged administration is necessary. The hypodermatic injection is more effective. From $\frac{1}{12}$ to $\frac{1}{6}$ of a grain of morphine is usually sufficient for an ordinary case, but, if the suffering be very great, $\frac{1}{4}$ to $\frac{1}{2}$ grain of morphine may be required. The combination of morphine and atropine is both more effective and safer, and hence atropine should be given, $\frac{1}{100}$ grain at each injection. Not only does this remedy remove the pain, but it is the most efficient means of preventing or subduing peritoneal inflammation. Anodynes can not be given by the stomach; anodyne enemata are insufficient in this malady—so that the choice of remedies is much restricted. Five minims of chloroform every half hour, in an emulsion or dropped on sugar, has been proposed, but in the author's experience it is usually rejected, and excites nausea even by its odor. It has been gravely proposed to administer it as a solvent of gall-stones, and to relieve the suffering by effecting a solution of the impacted calculus. Trousseau had, it was supposed, disposed of this notion, but it has been revived again. Chloral has also been employed to relieve the pain, but it is very offensive to the stomach; yet, if given by the rectum, it is highly efficient in relieving the pain by allaying spasm, and also, probably, has some solvent action. Warm baths and hot fomentations to the right hypochondrium contribute to relief. Undoubted advantage is derived from the use of leeches, when, the symptoms persisting, tenderness develops and fever arises.

Prophylaxis is highly important. The author has had abundant and highly favorable experience with the plan which is about to be recommended, and he therefore urges it on the attention of his readers: The diet must be carefully regulated. All fats and articles containing fat in any form are rigorously excluded. Saccharine substances are also prohibited, and the starchy constituents of the diet are reduced to a little white or corn bread—potatoes, beans, peas, and rice being excluded. Lean meat of all kinds, eggs, fish, fruit, and the succulent vegetables are permitted freely. Wine at dinner is allowed, but malt liquors and spirits are forbidden. Daily exercise is directed. All irregularities of life of every kind are given up. The most effective remedy for the removal of the conditions which lead to the formation of biliary calculi, or to bring about their solution, is sodium phosphate. This is prescribed in the dose of a drachm three times a day, dissolved in sufficient hot water, and taken before meals. This remedy is continued for several weeks or months, and, if there are present evidences of gastro-intestinal catarrh, $\frac{1}{20}$ of a grain of the arseniate of soda is added to each dose of the phosphate. While success seems always to attend this practice, the author has been constantly disappointed in the remedy of Durande (ether and turpentine), and in the administration of chloroform, with a view to its solvent action on retained calculi. As the catarrhal state of the bile-ducts, succeeding to catarrh of the duodenum, is the great factor in the causation of gall-stones, it is highly important to correct it. Without attention to the plan of diet above indicated this can not be accomplished ; but the persistent use of phosphate of soda can do much, even without a change in the habits of life, toward bringing about a cure. Vichy-water, and our own Saratoga Vichy, as well as the alkaline waters of this country, which are so abundant, should be used daily in connection with the plan above indicated. Dr. T. H. Buckler, of Baltimore, strongly recommends the use of the hydrated succinate of the peroxide of iron (℥ jss — ℥ vjss water —a teaspoonful *ter in die*) as a remedy to prevent the formation of calculi. The use of this remedy is based on some theoretical notions respecting the oxidizing power of succinic acid and its solvent action. Buckler also urges the use of chloroform during the paroxysms of colic, as a solvent of cholesterin. Harley advocates the use of sulphate of soda. Cholate of soda—a constituent of normal bile—theoretically an appropriate remedy, has been lately given with success, in doses of from two to five grains.

DISEASES OF THE SPLEEN.

TOPOGRAPHY OF THE SPLEEN.

In the normal condition of the organ the spleen is too small and too deeply placed to be felt, or indeed to be defined, even by careful percussion. Lying under the concavity of the diaphragm, covered by the stomach and by the transverse colon, and surrounded by the ribs, it is not brought within the range of our clinical methods. It is liable to displacement, the vessels forming a pedicle of less or greater length, permitting sometimes an extensive range of motion. Under such circumstances, instead of its natural position in the left hypochondrium, the spleen may glide downward into the umbilical, even as low as the hypogastric region. When enlarging from its true position in the hypochondrium, the change consists in an expansion, obliquely downward, of the area of splenic dullness.

The size and density of the spleen may be ascertained with an approximation to accuracy, by percussing the organ when forced downward and held by a full, retained inspiration, and comparison with the same area emptied by a full expiration.

The spleen being a highly elastic body, and its dimensions varying with the amount of blood contained in the vessels, there are rapid changes in the area of splenic dullness, strictly within physiological limits. Certain remedies, as ergot and quinine, have the power to contract the organ, but it is probable that this action only takes place when the spleen has undergone a pathological enlargement.

ACUTE SPLENITIS.

Definition.—By the term acute splenitis is meant acute inflammation of the spleen. Perisplenitis is a designation applied to inflammation of the investing tunic or capsule, and of the peritoneal layer of the organ. Acute splenic tumor means an acute enlargement—a condition present in various acute infectious diseases.

Causes.—Our present knowledge of the etiology of spleen-diseases is very unsatisfactory. Hardly anything is known of idiopathic splenitis. Of the secondary, or metastatic malady, our information, if not full, at least contains some certain data. That splenitis arises from embolism is now well known. Inflammation of neighboring parts extends to and involves the spleen. Direct injury, as a blow over the left hypochondrium, may excite inflammation in the spleen. A case arising in this way the author had under observation during life, and was present at the autopsy; hence the account given of the disease in question is derived largely from this experience.

Pathological Anatomy.—Local, or circumscribed, splenitis is induced by embolic blocking of a vessel or vessels, and hence the infarctions may be one, or two, or three in number; they may be in the substance, or at the periphery of the organ.* These infarctions vary in size from a pea to a hen's-egg, are wedge-shaped, and when near together may coalesce. These infarctions undergo the usual transformation, and a purulent collection is the ultimate result of the changes. A limiting membrane may form, and the pus become encapsulated, or the boundaries of the purulent depot may be constituted of the ragged, disintegrating, soft, splenic pulp. The pus tends to make its way externally, and when the capsule is reached adhesions form, usually to the diaphragm. In the author's case, as a result of a powerful blow on the left hypochondrium (which, however, left no external trace of the injury), the whole organ was turned into a brownish purulent collection of eighteen ounces' capacity. Adhesions had been formed with the diaphragm, which was softening, and adhesion of the opposed pleural surfaces indicated the preparation for discharge by a bronchus. The abscess may break into the peritoneal cavity, with the effect of inducing fatal peritonitis.

Symptoms.—As the systematic writers are not agreed as to the character of the symptomatology, the author describes it wholly from his own observation. After the injury, or we may also suppose the embolic obstruction, in a day or two, pain is experienced, deeply in the right hypochondrium. The sensation is rather of an aching character, which becomes soreness and tenderness when the organ is compressed—a feat that is accomplished by pressing upward under the ribs when the patient takes a full inspiration. There is usually pain developed by taking a deep breath, which becomes catching and acute when the peritoneum is invaded. Neither on palpation nor on percussion can an increase in the volume of the spleen be made out with certainty. In about a week after the initial symptoms, a rigor occurred, followed by fever and sweats, and these appeared irregularly up to the end. The face was pallid, the lips white, the sclerotic glistening, the body emaciated, and the weakness extreme. The appetite was lost, there was occasional vomiting, and diarrhœa supervened toward the termination of the case. Presently a harassing, dry cough, accompanied with pain and an obstinate hiccough, made its appearance. An increase in the left side through the hypochondrium and an enlargement of the area of splenic dullness now became evident. Death occurred by exhaustion on the forty-second day from the first symptoms.

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* Billroth; Virchow's "Archiv," Band xxiii, p. 473; "Der haemorrhagische Infarkt und seine Metamorphosen."

Course, Duration, and Termination.—Nothing can be more ill-defined than the course of splenitis. The duration of cases of inflammation terminating in abscess may be not more than a month, and yet cases have continued several years (Mosler). Splenitis may terminate in resolution without symptoms. This is the most probable explanation of the existence of cicatricial depressions on the surface of the spleen, found in cases dying from other causes. Cases proceeding to suppuration terminate by discharge through the lungs, of which a successful case has been reported, or communication is established with the stomach, the transverse colon, the left kidney, or with the general cavity of the abdomen.

Diagnosis.—If endocardial lesions exist, and sudden pain followed by swelling occur in the splenic region, and subsequently there arise the usual symptoms of suppuration, or if, as a result of a blow, pain and tenderness and swelling develop in the left hypochondrium, the spleen may be presumed to be the seat of the mischief.

Prognosis.—As those cases of splenitis which terminate in recovery are never recognized, the question of prognosis does not come up for solution. When abscess occurs, the prognosis is unfavorable.

Treatment.—If the existence of splenitis, from any cause, is ascertained, quinia must be freely administered, and cinchonism maintained. There are two good reasons for this practice : quinia checks the migration of the white corpuscles and the process of suppuration, and lessens hyperæmia of the spleen. No therapeutical fact is better established than that quinia reduces the size of the spleen when it is enlarged by hyperæmia. Quinia is, therefore, peculiarly adapted to the treatment of splenitis. Purgatives act on the spleen in two modes ; by reflex action, and by diminishing the general blood-pressure. Saline cathartics should be used to maintain free action of the intestines. Warm fomentations, turpentine-stupes, and hot poultices should be applied over the left hypochondrium. If suppuration is clearly ascertained, the aspirator should be used without delay, just as it is now employed in a similar state of things in the liver. The strength must be kept up by suitable food and stimulants.

ENLARGEMENT OF THE SPLEEN.—Owing to its peculiar anatomical structure, the spleen is especially liable to variations in size, strictly within physiological limits. In the acute infectious maladies the organ undergoes a change in size of a pathological character. In typhus, typhoid, puerperal, and the eruptive fevers, the spleen enlarges, but in the fevers of marsh-miasm the change in size is greater.

In certain parts of this country—the Wabash Valley, for example—a splenic tumor of extraordinary size (ague-cake) sometimes develops under the influence of malaria without the objective phenomena of fever, but with the same bodily changes as occur in intermittent and remittent fevers. Obstructive diseases of the heart, lungs, or liver, by causing stasis in the venous system, give rise to enlargement of the spleen, and especially does this result follow sclerosis, and acute yellow atrophy of the liver. In the condition of enlargement which occurs during the course of fevers—excepting from consideration malarial fevers—the spleen is excessively soft, the splenic pulp almost diffluent, the capsule and trabeculæ easily torn. In the acute enlargement which accompanies the febrile movement of malarial fevers, there is really no alteration of structure—the pulp and trabeculæ and the Malpighian bodies having their normal appearance and structure, but the increase is due to an immense venous congestion. On the other hand, in the enlargement which occurs without fever, or produced after successive attacks of fever, the organ is dense, firm, and paler, due to the great development of the trabeculæ and corresponding diminution of the splenic pulp. In these cases of chronic enlargement due to malarial infection, the organ may attain considerable size, greatly distend the abdomen, and reach to and even extend beyond the umbilicus. There is in these cases an extreme anæmia—a pseudo-leukemia—the superficial veins of the abdomen are enlarged, the legs are swollen, and there is some effusion in the abdomen—results of the mechanical pressure. A splenic tumor of medium size, formed in the mode above indicated, may lodge on the aorta and be confounded with aneurism.

MISPLACEMENT OF THE SPLEEN, or MOVABLE SPLEEN.—Changes in the position of the spleen are effected by effusions in the left thoracic cavity, which displace the organ downward. When enlarged and in the condition of "fleshy spleen" above described, the spleen may descend considerably by its own weight, and thus seem more enlarged than it is really. The movable spleen, like the movable kidney, is displaced from its position, and its vessels with the omentum are stretched and ultimately assume the shape of a pedicle—an irregularly rounded cord—of which the author has seen several capital examples. Such a spleen may be moved by a change in the position of the patient, or by palpation, and may lie across the abdominal artery and be lifted up synchronously with the arterial pulsation, or be displaced downward into the iliac fossa, and may rotate on its horizontal axis. Changes in the structure of the organ necessarily occur under these circumstances; the blood-supply is lessened, or thromboses form in the vessels; there are shriveling and atrophy, pigmentary and fatty degeneration, etc.

AMYLOID DEGENERATION OF THE SPLEEN.—This disease consists in the deposits of the amyloid matter, either in the form of small patches, forming the well-known "sago-spleen," or in a general diffusion of the material through the whole organ. In the former the patches may be very numerous and almost unite, but there still remains normal spleen-tissue between them. In the latter form the texture of the spleen is firm and tough, but easily divided with the knife, although not readily broken up into a pulp, and it has a brownish or yellowish-brown color, and no part remains untouched by the new deposit—the pulp, the trabeculæ, the Malpighian bodies, the vessels, all are changed in structure and physical properties by the amyloid matter. The test for this matter is iodine—Lugol's solution—which when brushed over colors the tissues yellowish, but the amyloid matter red or reddish brown : now, on the addition of sulphuric acid, while the yellowish parts remain yellow, the amyloid becomes a dark violet. The amyloid, or lardaceous, or waxy degeneration of the spleen occurs, simultaneously with the same form of degeneration in the liver and intestinal canal, and hence the symptomatology is rather that of the disturbance in the function of the other organs. These symptoms have been detailed in the remarks on amyloid liver. The only contribution made to the symptomatology by the alterations in the spleen are, the increased area of splenic dullness and a greater degree of anæmia and pseudo-leukemia. The great cause of amyloid degeneration of the spleen as of other organs is suppuration, especially protracted suppuration in connection with bone. Next to this are the syphilitic cachexia and inherited syphilis. Chronic alcoholism and chronic malarial poisoning are supposed to have some influence in its production, but it is extremely doubtful whether they have any real influence.

ECHINOCOCCUS OF THE SPLEEN.—The embryo of the tænia echinococcus is deposited in the spleen as in other organs, and more frequently in the spleen than in any, except probably the liver. The liver is reached readily by the portal vein, and the spleen directly, as the two organs come into contact. When established in its home, growth begins, chiefly by the development of daughter-vesicles in the mother-sac. The symptoms produced are due to the size to which the sac attains, the pressure on neighboring organs, and the interference with the circulation in the great vessels of the abdomen. The slowness of the growth, the absence of constitutional disturbance, the freedom from pain, and the absence of symptoms except those due to the size of the tumor, separate the echinococcus from other tumors of the spleen. The sense of fluctuation, and especially the purring tremor, serves to distinguish this from hypertrophy of the spleen. The employment of the aspirator-needle will contribute to certainty of diagnosis, but the presence of hooklets and the absence of albumen can not always be depended on, for the hooklets may be absent, and albumen may be present in echinococcus tumors of the spleen. For further details the reader is referred to the subject of echinococcus of the liver.

DISEASES OF THE BLOOD-FORMING ORGANS.

THE BLOOD.

It will facilitate the comprehension of the maladies treated of in this section, to precede the account of the several morbid states with some observations on the nature and composition of the blood.

Composition.—The ultimate chemical composition of the blood is as follows :[*]

Reaction	alkaline
Specific gravity, from	1,045 to 1,075
Water	781·6
Globules	135·
Albumen	70·
Fibrin	2·5
Fats	1·5
Extractive matters	2·4
Salts	6·5
Iron	·5

The results of chemists vary according to the method of analysis employed. The most considerable departure from the ordinary method is that of Flint, who tries to measure the several constituents in their natural state ; that is, with their water of composition and the inorganic salts so closely associated with them as to be separated only by incineration.[†] There is much to be said in favor of this method. Without engaging in this discussion, the several constituents of the blood will be briefly considered, with reference to the changes induced by disease.

In general terms, the blood may be considered as made up of certain morphotic or formed constituents—the corpuscles, red and white —floating in a complex fluid, containing, dissolved in it, albumen, fibrin, fats, and salts. The red corpuscle consists of two parts : the matrix, or stroma, which seems only to serve the purpose of a vehicle for its active constituent, the *hæmoglobin*, the other part. The most important function of hæmoglobin is its power to form compounds with gases of a very unstable nature, the chief being the combination with oxygen, *oxy-hæmoglobin*, or *oxide-hæmoglobin*. The oxygen is readily taken up and as readily parted with, this process constituting the so-called ozonizing or respiratory function of the blood. The arterial blood contains 16·9 per cent. in volume of oxygen; whereas the venous blood only has 5·96 per cent. Now, as this process of oxidation and deoxidation of the hæmoglobin goes on incessantly, the importance of the function is obvious. Hæmoglobin is soluble in water, and crystallizes from its solution in fine crystals, called "blood-crystals."

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[*] Becquerel et Rodier, "Traité de Chimie Pathologique Appliquée à la Médecine Pratique," etc.

[†] The "Physiology of Man," vol. i, p. 133.

The white corpuscles, leucocytes, contain a good deal of protoplasm, and are closely allied to lymph and pus-corpuscles. They are derived from the lymphatic glands and lymph, and have a power of motion, which is called amœboid, in virtue of which they may migrate from the vessels into the surrounding textures.

The relative proportion of red and white corpuscles varies considerably within physiological limits. The average of the red corpuscles is in the proportion of five millions in a cubic millimetre of blood, and the white bears the proportion to the red of one to 800, and after a meal of one to 300 or 400. These figures must be regarded as merely approximative. The red corpuscles "constitute about one third of the weight of the blood, and contain about 43 per cent. of solids, and 39 per cent. of hæmoglobin" (Burdon-Sanderson).

The fluid in which the corpuscles are suspended is the *plasma*. Serum is the plasma which has lost its plasmin by coagulation. When blood is allowed to stand under suitable conditions, it separates into corpuscles and plasma. At ordinary temperature, plasma coagulates, and a clot forms, composed of fibrin, with the corpuscles entangled in its meshes. This fibrin-clot when in its dissolved state is *plasmin*. The coagulation of blood consists in the solidification in delicate filaments of the plasmin, which when it assumes this form becomes fibrin. Besides plasmin, which is an albuminous substance, there are two albumens in plasma—ordinary albumen, and a small quantity of alkali-albumen. The serum, and coagulable liquids generally, contain *paraglobulin* and *fibrinogen*, but the action of a third substance is necessary to coagulation. This is a *ferment*, now supposed to be produced by the white corpuscles. When these substances are present, spontaneous coagulation will take place.

Blood plasma contains, also, 0·5 per cent. of the solid salts of the blood, consisting, for the most part, of sodic chloride, sodic phosphate, and sodic carbonates (chloride, phosphate, and carbonate of sodium).

Examination of the Blood.—Apply a suitable ligature to the finger, make a puncture with a lancet, and receive a drop of blood on a perfectly clean glass slide warmed to 100° Fahr.; cover at once with thin glass, which has been moistened by breathing on, and gently press it down until there is a stratum of blood so thin as hardly to appear red. It can now, and as quickly as possible, be examined with a quarter-inch objective. The red globules are particularly well seen with the binocular of Beck. The condition of the red corpuscles, of the white, and their relative numbers, should be noted. The action of a reagent may be watched by bringing it to the margin of the cover-glass, when it will gradually diffuse into the blood. Besides the ordinary constituents of the blood, there may be seen, under peculiar circumstances, certain parasites, pigment granules and cells, crystals of uric acid, etc.

The Hæmacytometer.—This instrument, as its name indicates, is intended to ascertain the number of blood-corpuscles. Devised in a crude form by Vierordt, afterward improved by Melassez and Hayem, the most convenient arrangement has been perfected by Gowers, and made by Hawksley, of London. The process of counting the corpuscles by means of the hæmacytometer consists essentially in dilution of the blood to a definite degree, and numbering the corpuscles contained in a cell of given depth on a slide marked with ruled lines which indicate the lateral dimensions of the dilution. In this way quite a close approximation to the true number of cells in a given measure of blood can be made. The morbid conditions of the blood to the diagnosis of which this method is applicable will be considered further on.

The Hæmoglobinometer.—The peculiar function of hæmoglobin is so essential to the work of the body that means for ascertaining its quantity are very desirable. The hæmoglobinometer is an instrument by which the proportion of hæmoglobin in the blood may be determined. Melassez and Hayem first demonstrated practicable methods, but the profession is here again indebted to the practical skill of Gowers for the perfection of an available apparatus. It consists, first, of a standardized solution of glycerin-jelly, colored to the tint of normal blood, and contained in a tube of given capacity; second, of a tube of the same capacity as the first, for the diluted blood under examination. The degree of dilution necessary to make the blood-tint correspond in depth to the standard indicates the proportion of hæmoglobin. Less accurate but more ready methods for approximating to the proportion of the blood coloring-matter are given in the section on *hæmoglobinuria.*

Pathological Relations of the Blood.—Before entering on the subject of the special pathology of the blood, it will be useful to indicate in outline the modes in which the blood is changed. The several morphotic elements may be altered quantitatively and qualitatively, and the constituents of the plasma may be increased or diminished, or entirely removed.

The red corpuscles of the blood diminish in number, and that to an extreme degree, so that the normal proportion of red and white may be reversed. Alterations in the shape of the corpuscles sometimes take place, but they have no precise signification, and have not thus far been studied with success. In the process of coagulation, the red corpuscles may not exhibit the normal behavior, and unite in *rouleaux*, but rather adhere in irregular masses. The significance of such a condition is not understood. New forms of corpuscles may appear, some of them transitional between the white and red.

The blood may assume the appearance known as "lake," and become transparent instead of opaque. Such a change as the term lake supposes, indicates disorganization of the red corpuscles, and the separation of the coloring matter, which dissolves in the plasma. Various changes of color may take place. The transformation of venous into arterial blood may not occur, and the whole mass of the blood continue of the venous hue. It may assume a chocolate tint, from the action of certain medicaments.

The plasma of the blood may be variously altered. The relative amount of water may be increased or diminished, or, as it should rather be stated, the proportion of solids is increased or diminished. This change may be effected by some medicaments, but is most pronounced in certain morbid states; for example, in the algid stage of cholera, when the loss of water from the blood, and its artificial replacement by intravenous injection, make a wonderful change in the condition of the subject. Variations in the proportion of albumens in the blood are caused by activity in the function of nutrition, and excess in the processes of waste and excretion. When albuminous foods are abundant, digestion active, and absorption prompt, the blood may contain relatively more albumen, but the excess above the needs of the organism will be deposited as fat, or excreted as uric acid and urea. Under some circumstances fibrin may be in greater than normal quantity, constituting the condition of *hyperinosis*, in which the coagulation of the blood is more prompt, and the clot firmer. Formerly the "buffy coat" was supposed to be indicative of hyperinosis, but this appearance is now known to be due rather to the condition of the red corpuscles, and their manner of settling in the process of coagulation.

The salts of the blood are subject to considerable variation under conditions not now understood. Salts of potassium base are contained in the corpuscles chiefly, while those of sodium base are to be found in the plasma. The normal reaction of the blood, which is alkaline, may be changed to neutral, even to acid, by chemical transformations in the salts.

The quantity of fat in the blood varies considerably within physiological limits. A diet rich in fat, the use of alcohol, obesity, tend to increase the proportion of fat in the plasma. The serum of the blood may, indeed, present a milky appearance in consequence of an excess in the number of fat-globules which have entered the vessels, and fat-embolisms are caused by the admission of fat-cells into torn vessels in some cases of fracture of bones.

Urea and uric acid are found in the blood in small quantity quite constantly, but in certain morbid states in excessive amounts. Crystalline forms of urate of soda may, in some cases of gout, be detected in the serum. Leucin, tyrosin, acetone, and bile-pigment are also constituents of the blood, to be found during the existence of certain morbid states.

To an expert the examination of a drop of blood is a comparatively easy task. It is only rarely that such an examination is necessary, when the indispensable skill is available. There is reason to fear that the routine examination of the blood, now so much spoken of, is rather intended to impress the patient than increase the knowledge of the conditions present.

LEUCOCYTHEMIA—LEUCÆMIA.

Definition.—The terms leucæmia and leucocythemia were proposed by rival claimants for priority of discovery—Virchow and Hughes Bennett. The term leucocythemia, proposed by Bennett, seems to the author a more correct designation, meaning *white-cell blood,* than Virchow's leucæmia, which means *white blood.* The morbid change which has given the name to the disease is the enormous increase of the white corpuscles of the blood, accompanied by enlarged spleen and enlarged lymphatic glands, and by alterations in the marrow of bones. By Trousseau it is designated *adénie,* and by Griesinger *anæmia splenica.*

Causes.—The excessive production of leucocytes, which is the chief element in this disease, must necessarily be due to a functional and nutritive irritation of the blood-making organs. The evidence of this is afforded in the enlargement of the spleen and lymphatic glands. But the cause of this remains unknown, and hence the real nature of the malady continues an insoluble problem. Leucocythemia occurs at all ages and under every kind of social circumstance, but it attacks by preference the male sex, the most vigorous period of life—thirty to forty-five—and those who have been weakened by hardships and excesses. Menstrual irregularities have been supposed to have an influence in developing it, and, in twenty-one cases of this disease occurring in women, there were sixteen in whom some disorders of the uterus had existed (Mosler). It is probable that these sexual irregularities were rather coincident than causal. The cachexiæ of chronic malarial poisoning and of syphilitic infection have been invoked to account for its production, but no satisfactory data have as yet been published, although there are examples of accidental association. Regarded from the analogical point of view, leucocythemia may be classed with scrofula, cancer, tubercle, and other infectious diseases, which, beginning at one point, or focus, diffuse thence over the body. The morbid alterations characteristic of this disease begin in the spleen, then attack the lymphatic glands, then the marrow of bones, and thus become general.

Morbid Anatomy.—The most constant lesion is in the spleen, which is increased in size, either uniformly, its form and shape being preserved, or some part of the organ undergoes the change. Not only the size but the firmness and density are increased. The color becomes a reddish blue; the pulp undergoes hypertrophy, but the normal relations of its elements are preserved; the trabeculæ may be more distinct, or may be obscured by the overgrown pulp; the Malpighian bodies are rather increased in number, very distinct, but less consistent than normal. The trabeculæ and pulp may be coated with a yellowish, fibrinous exudation; there may be seen white granules disseminated throughout the organ, and near the surface patches of indurated tissue, the remains of hemorrhagic infarctions. The change in the lymphatics consists in an initial hyperæmia, then hyperplasia of its constituent parts, first of the cellular elements, then of the stroma and vessels. They enlarge in proportion to the addition of new material, from a bird's egg to a goose-egg or larger. They have a smooth, rather glistening, appearance, and to the touch are soft, non-elastic, and sometimes fluctuating. All of the lymphatic glands in the body may be engaged, or the process may be confined to a few. Usually those situated about the hilus of the liver and spleen are enlarged. Similar changes take place in the lymphatics of the digestive tract, beginning in the follicles of the tongue and tonsils, of the stomach, and in the glands of Peyer. Corresponding changes occur in the marrow of long bones, and in the cancellated tissue of the ribs and sternum. The marrow is abundantly infiltrated with lymphoid cells, and the vascular network with its delicate connective tissue, which exists in the normal condition, disappears, and only the larger arterial branches remain. The result is that the marrow, instead of its rose-color, becomes yellowish or greenish yellow.* In somewhat more than one half of the cases the liver is enlarged and changed in structure by reason of the development of the new lymphadenoid tissue of the organ. It increases in size, sometimes immensely so, and weighs from four to eighteen pounds. This change is at first a mere proliferation of the lymph-cells; then occurs an infiltration of lymph new formations, or these are collected in masses or nodules, like tubercle. The cells penetrate the lobules from without inward, and by their numbers dispossess the hepatic cells, which atrophy and disappear, only spots of pigment remaining.† The most important change is that which gives the name to the disease, the increase of white cells in the blood.

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* Mosler, *op. cit.*

† Rindfleisch, "Pathological Histology," pp. 183, 473, American edition.

The gross amount of blood is not lessened, but its specific gravity is reduced from 1055 to 1040, even to 1035.* The color is paler than normal, and purulent looking. The proportion of white corpuscles is relatively greatly increased; but the numbers vary from one to ten, to one to two; indeed, the white and the red may be equal in numbers; the white may even preponderate. The white corpuscles may differ from the normal in being larger; in splenic leucocythemia they contain one or several nuclei; sometimes the cells are smaller, and there is one large nucleus; and occasionally transitional forms are discovered between the white and red, such as are found in the cell-masses of the marrow.† The red corpuscles are both relatively and absolutely diminished in numbers, the water and fibrin are increased, the iron diminished, and certain abnormal ingredients are present, as formic, lactic, and acetic acids, hypoxanthin, uric acid, leucin, tyrosin; but, of these, lactic and formic acids and hypoxanthin only are constantly present (Mosler). According to the same authority, the reaction of the blood in this disease is not acid, but alkaline. The morbid processes of leucocythemia are not those of a merely splenic disease—a local malady. Hyperplasia of the spleen is, however, the first link in the chain; from this organ, immense numbers of leucocytes pour into the blood, and also, it is probable, some products of the splenic pulp, as lactic and formic acid, and hypoxanthin, etc.; the next step consists in the transplantation and subsequent development of heteroplastic materials in other organs, as the liver, etc.

Symptoms.—According to the preponderance of the leucæmic process in the spleen, lymphatics, or marrow of bones, the disease is entitled splenic leucocythemia, lymphatic leucocythemia, and myelogenic leucocythemia—for these organs seem equally to possess the power of producing white corpuscles and introducing them into the blood, and one may perform the office for the others. When the spleen is removed there are very few defined disturbances of the functions, as the lymphatics and the marrow of bone perform the necessary offices. It is the splenic form of the disease which is usually encountered, or the splenic-lymphatic, and the lymphatic very rarely, and the myelogenic never. The development of leucocythemia is so gradual that the beginning of symptoms usually passes unnoticed, unless preceded by syphilitic or other lesions, to which attention has been directed. There is usually a history of the gradual appearance of weakness and anæmia, inability for mental and especially for any physical exertion, headache, ringing in the ears, vertigo, palpitation. There are, as the anæmia gradually develops, alternations of an improved state with more decided decline, but the constant tendency is downward. These prodromal symptoms last from a few months to several years, the average being about eighteen months. As the cases progress, the condition of anæmia becomes more profound ; the lymphatics of the neck, or groin, or other superficial parts, are found to be somewhat enlarged, and now careful palpation discloses enlargement of the spleen. There are, then, extreme pallor, weakness and exhaustion, and breathlessness on the slightest exertion. The headache, vertigo, and tinnitus continue, and the mental state is depressed, hypochondriacal, and irritable, "due to the accumulation of white corpuscles in the capillary vessels of the brain."* The vision is obscure and amblyopic. There are now and then, without apparent cause, attacks of profuse sweating, and scaly and pustular eruptions. There is usually some feverishness toward evening, and the pulse is always accelerated. Œdema of the ankles, puffiness of the eyelids, and some effusion in the cavities are results of the hydræmia. The changed condition of the blood also induces the hæmorrhagic cachexia or diathesis, and bleeding occurs from the nose, mouth, and other mucous surfaces, and from slight wounds, so that the least abrasion or cut gives rise to severe hæmorrhage. The vessels remain unaffected except by capillary thromboses, due to the aggregation and adhesion of white cells, and such changes in their walls as are produced by imperfect nutrition. A soft-blowing murmur—anæmic murmur—is audible at the base of the heart. The appearance of the blood is very characteristic. A ready method of demonstrating its character has been mentioned by Sir William W. Gull †—that is, "puncture the finger of the patient, and receive the blood on to a piece of white linen, or a lawn handkerchief, and put by the side of it a similar stain of blood from a healthy subject. The full color of the latter contrasts strikingly with the stain of the former, which is hardly of a blood-color, and translucent." The relative proportion of blood-globules is best ascertained by counting, employing for this purpose the hæmacytometer as arranged by Gowers. In order to constitute leucocythemia, it has been attempted to fix arbitrary numbers, but, while the proportion of white to red corpuscles must be increased very largely above the normal, yet no definite number can be stated, and hence the diagnosis must rest rather on the concurrence of the splenic and lymphatic enlargements with increase of the white corpuscles. It may, however, be stated, as an approximation to the truth, that the relative proportion of white to red should be reduced to one to six, in order to constitute true leucocythemia. It has already been stated to what extent the disproportion may be carried in this disease when fully established. When the spleen has reached its maximum, the abdomen is greatly enlarged, and prominent, but in ordinary cases an increase of size, and usually of density, can be ascertained on palpation. The mesenteric glands can usually be felt through the abdominal walls, enlarged and firmer. The inguinal, cervical, and other lymphatic glands, are also enlarged. A capital illustration of these is given in the plate accompanying Surgeon-Major Porter's case,* as reported to the London Pathological Society.

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* Wagner, *op. cit.*, p. 546.

† Renaut, "Archives de Physiologie," September, 1881.

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* Ollivier et Ranvier, "Nouvelles Observations pour servir à l'Histoire de la Leucocythémie ;" "Archives de Physiologie," vol. ii, 1869, p. 518.

† "Transactions of the Pathological Society," vol. xxix, 1878, p. 383.

The tumors of the tongue and tonsils interfere with mastication and the act of swallowing; the gums become spongy and tender. The appetite may be keen; it may be normal; it may be wanting entirely. Constipation at first is present; then diarrhœa alternates with constipation, and finally diarrhœa persists. The urine has a higher specific gravity than normal—from 1020 to 1030. The urea is greatly diminished, but the uric acid is increased, and hypoxanthin is present, in the cases of splenic leucocythemia.

Course, Duration, and Termination.—Leucocythemia is essentially a chronic malady. Its origin can not be often determined, because there is a slow development of uneasiness in the splenic region, fullness of the abdomen, breathlessness on exertion, and anæmia and pallor of the skin. The swelling of the spleen, until its size is considerable, escapes recognition; when, however, the external lymphatic glands enlarge, attention is earlier directed to the nature of the case. Then an examination of the blood furnishes conclusive evidence. When the hæmorrhagic diathesis comes on, bleeding may be so severe as to exhaust the patient rapidly, or death may occur suddenly by cerebral hæmorrhage. The course and duration of cases are materially affected by the hæmorrhagic diathesis. When this does not exist, the progress is much slower and the duration more prolonged. The glandular and splenic enlargements may become enormous, and the patient die ultimately of exhaustion, death being preceded by cerebral symptoms—delirium, stupor, and insensibility. The case may be terminated by some intercurrent malady, as pericarditis, pleuritis, pneumonia, etc. The symptoms of the first stage, as already stated, continue for months, even years, the average being about eighteen months, and the second stage, or fully developed malady, lasting about one year. Probably the average duration of the whole disease is two years.

Diagnosis.—In the first stage of this malady a distinction is not possible from ordinary anæmia and chlorosis. When, however, the spleen enlarges, and the lymphatic glands also, and the anæmia becomes extreme, the picture of the disease is complete, and no one possessed of any knowledge could fail to recognize it. In the early stage, the persistence of the anæmia under appropriate treatment, the extreme degree of pallor, the breathlessness under slight exertion, the vertiginous sensations, the hæmorrhagic diathesis, must awaken suspicion as to the character of the malady, before the splenic disease manifests itself.

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* "Transactions of the Pathological Society," vol. xxix, p. 339, *op. cit.*

Treatment.—Unfortunately, we possess no specific against this disease, and hence the treatment must be symptomatic. Iron, which is a specific in anæmia, has no influence of a curative kind in leucocythemia, but it is useful as supplying a material needed in the process of repair. There are several remedies which affect the spleen, in a way which indicates a specificity of action: they are quinia, ergotin, and electricity. Quinia, iron, and ergotin can be given together in pill-form —five grains of quinia, one grain of reduced iron, and two grains of ergotin, should be administered three times a day. Simultaneously, electricity can be applied in the form of faradic electricity to the splenic region, or by means of an insulated electrode in the rectum, and the other over the spleen. A slowly interrupted galvanic current is, the author believes, more efficient. Good results are obtained from the local application of the ointment of the biniodide of mercury—unguentum hydrargyri iodidi rubri—to the splenic region. The ointment should be thoroughly rubbed in while the direct rays of the sun are falling on the part, or before a bright fire. The ointment is rubbed in daily, until the skin begins to vesicate, when it must be discontinued, but resumed again when the skin has recovered from the effects of previous applications. As the breathlessness on exertion, the vertigo, the mental troubles, the effusions, the hæmorrhages, etc., are due to the impoverished blood, attention must be directed to the central lesion, rather than administer remedies for individual symptoms. In some cases good results have apparently followed tranfusion of blood; but they were examples of the hæmorrhagic diathesis, rather than of true lencocythemia. In the latter disease transfusion is useless—three cases in which it was employed by Stoll, of Wurzburg, having proved fatal. As the function of blood-production is at fault, attention to the first steps in the process is necessary: in other words, careful alimentation is of great importance. Whether the appetite be languid or voracious, to insure thorough digestion, pepsin and muriatic acid should be administered after each meal. As, in the progress of the disease, the liver and intestinal glandular apparatus are disabled, fats, starches, and sugars should be excluded from the diet as far as possible, and the patient be fed on fresh meats, milk, eggs, and fish. Cases not yielding to the plan above indicated may be treated with arsenic, arseniate of iron, especially Fowler's solution, and the phosphates or compound sirup of the hypophosphites. These remedies should, of course, be pushed, especially the phosphates, for no immediate results can be obtained from them. Arsenic has been administered hypodermatically, and injected directly into the substance of the enlarged spleen with asserted advantage.

ADDISON'S DISEASE—MELASMA SUPRARENALE.

Definition.—Dr. Addison, of Guy's Hospital, in London, in 1855 announced the discovery of a disease in which, with a peculiar bronze-like discoloration of the skin, there are associated great weakness and anæmia, the whole being due to disease of the supra-renal bodies. His name has, by common consent, been associated with this disease permanently, which is hence known as *Addison's Disease—Morbus Addisonii.* It is sometimes called "the bronzed-skin disease," "melasma suprarenale," etc. It may be defined, in the words of Averbeck,* as "a well-marked constitutional disease, exhibiting itself locally as a chronic inflammation of the supra-renal capsules, but in its essence consisting in a peculiar anæmic condition, always tending toward death, which is characterized by intense development of pigment in the cells of the rete Malpighii and in the epithelium of the mucous membrane of the mouth."

Causes.—Although the anatomical structure of the supra-renal bodies has been successfully studied, the knowledge of their physiological functions has not advanced correspondingly. It is, therefore, difficult, even impossible, to trace a relationship between symptoms of the disease and some observed lesion. It occurs rather more frequently among men, and is a disease of adult life, no case occurring before ten or after sixty years of age. Dr. Greenhow † has collected one hundred and nineteen cases in males and sixty-four in females, of whom eleven years was the youngest, and fifty-nine the oldest, age at which death occurred. There is, however, an exceptional case in which the limit of maximum age is exceeded—that of a woman who is reported to have died at sixty-nine. Various depressing moral emotions and evil hygienic influences have been assigned a causal relation to the disease. Grief, anxiety, fear, exposure to cold and dampness, and want, are supposed exciting causes. A considerable proportion of the cases have coexisted with tuberculosis in other organs, and a few well-marked examples of the disease have been apparently due to a tubercular degeneration of the supra-renal bodies; hence the strumous constitution or diathesis is supposed to be intimately related to the disease. In Averbeck's collection of fifty-one cases, there were evidences of tubercular, strumous, or caseous degeneration in thirty-six. Greenhow holds that the condition of general tuberculosis, without being necessarily causative, frequently coexists with Addison's disease, and that "in a certain small number of cases the genuine lesion in the capsule has been found coexisting with advanced phthisis or general tuberculosis." It seems clearly established that Addison's disease is frequently associated with inflammation and suppuration in neighboring structures—extending to and ultimately involving the supra-renal bodies. Caries of the vertebra, psoas and perinephritic abscess, etc., are examples of such causes. Greenhow insists on traumatism as an influential factor, but this must be regarded as an exciting cause. A considerable proportion of cases occur without any obvious reason for their appearance.

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* "Die Addison'sche Krankheit," Erlangen, 1869. (Merkel.)

† "The Croonian Lectures for 1875," "The Lancet," vol. i, 1875.

Pathological Anatomy.—The condition of the supra-renal bodies varies with the period of the disease at which the examination is made. Virchow* describes a cheesy degeneration, beginning in the medullary tissue as small gray nodules, which gradually increase in size, amalgamate, and ultimately become caseous. A portion of the body may remain normal, or, gray nodules forming also in the cortical tissue, the whole organ may finally degenerate into a firm, caseous mass. The English authorities, especially Wilks and Greenhow, maintain that the disease in the supra-renal bodies is a low form of inflammation, during the course of which these organs become infiltrated with a fibrous exudation that undergoes conversion into a purulent, caseous, or cretaceous material. According to Merkel, the process is essentially tubercular. The nerves having intimate relations with the supra-renal bodies are also affected by a low grade of inflammation, consisting in congestion of the neurilemma, hyperplasia of the connective tissue, atrophy, fatty degeneration, and pigmentation of the ganglion-cells. The capsules which are in close proximity to the solar plexus and semi-lunar ganglia are more abundantly supplied with nerves, relatively to their size, than any organs in the body. The cells of the medulla present a strong resemblance to the multipolar ganglion-cells of the brain and spinal cord, and a number of nerve-trunks pass through the cortical portion into the medulla (Leydig). From this anatomical disposition it is maintained that the medulla is a portion of the ganglionic nervous system, while the cortex is a vascular gland (Kölliker). The nerve-trunks are invaded by contiguity by the inflammation attacking the capsules. The fibers and ganglia of the solar plexus are also changed. The neuritis, beginning in the nerves of the supra-renal bodies, extends to the fibers of the plexus and to the semilunar ganglia, the cells of which become cloudy and granular and pigmented, with subsequent atrophy. Traces of old and recent hæmorrhages are also found.

The blood is by some said to be deficient in fibrin and to have an excess of white globules, and the red globules do not manifest any tendency to form *rouleaux*. Greenhow finds that the blood does not exhibit any appreciable alteration in typical cases, and that the changes observed by Buhl and others were due to coincident but accidental lesions in the lymphatic glands. Atrophy and fatty degeneration of the heart and of the intima of the vessels have been observed in some

* "Krankhaften Geschwülste," Band ii, p. 689.

cases. The urine is deficient in urea and in pigment, and contains an excess of indican. The most striking change during life—the abnormal pigmentation—is due to the deposition of granular pigment in the cells of the rete Malpighii, in the papillary portion of the cutis, and even in the connective-tissue corpuscles. No change occurs in the proper structure of the skin. Similar pigment deposits occur in the mucous membrane of the mouth, especially along the edges of the teeth, while the conjunctiva, the nails, and the skin of the palms and soles of the feet are entirely free from deposits. It is obvious that the phenomena of Addison's disease—the anæmia, the feebleness, and the bronzing of the skin—are due to changes in the supra-renal bodies or in the connected nerves or ganglia. That alteration of the capsules will produce such symptoms can not be admitted, in view of the fact that cancer and other forms of degeneration have destroyed these bodies without causing this malady. It is the peculiar lesion of the supra-renal capsules, of the connected nerves, and of the solar plexus, with the semilunar ganglion only, that really produces the morbid complexus of Addison's disease. Unquestionably the changes in the nerves and ganglia are the most important pathological factors, and to them are ascribed the strange constitutional symptoms, including the pigmentation, by the chief authorities who have discussed the pathology of this malady. The view that the abnormal pigmentation occurs through the medium of the nervous system is supported by the fact that the discoloration lessens with improvement in the constitutional state, and deepens and extends when the symptoms indicate a more active condition. Analogous influence of the nervous system is seen in those cases in which a permanent darkening of the skin has followed sudden fear and agony of mind (Greenhow). It is the trophic system, especially, which is concerned. Doubtless Jaccoud nearly expresses the correct pathology when he holds that in Addison's disease the changes in the supra-renal bodies excite an irritation of the vaso-motor (trophic ?) system, which requires a much longer time to produce the pigmentation than to develop the asthenia ; hence the long existence of the latter before the appearance of the former.

Symptoms.—Addison's disease develops very gradually—so gradually that the time of beginning escapes observation. First, an unwonted sense of weariness on exertion is experienced. The debility slowly, in some exceptional cases quickly, increases, until a marked degree of asthenia is reached. There is not a corresponding degree of anæmia and wasting, and, although the sclerotic is pearly, the mucous membrane is red, and the blood has the normal proportion of red globules, while the subcutaneous fat is little if at all diminished, the muscles being weak and flabby. Disorders of digestion succeed to the early symptoms of debility. An unpleasant distention of the stomach is felt after eating ; eructations, nausea, and occasionally vomiting occur; and during, sometimes between, the acts of digestion, considerable pain is felt in the stomach. Pains of a dragging, tearing character also occur in the hypochondria and in the spine, extending into the sacrum. Much tenderness is elicited by pressure in the hypochondria, especially in the right, where, besides, the pain is most acute. The joints also become the seat of considerable pain, which is increased by movement, but they are neither swollen nor tender. These pains are pretty constant, while the disorders of digestion are at first intermittent, becoming more and more frequent as the case progresses.

With the development of these symptoms the asthenia increases. The least exertion induces an overpowering sense of fatigue. An extreme pallor of the skin—of those parts unaffected by pigment deposits—and a weak, soft, extremely small pulse, indicate the failing circulation. The impulse of the heart grows weaker and weaker, and a soft blowing murmur is audible at the base and over the course of the great vessels. So exceedingly weak does the heart become, that the patient, when still able to sit up, may have no pulse at the wrist. The skin is cool and the temperature is below normal, although, under exceptional circumstances, an evening rise to febrile heat may be observed. Meanwhile, the gastro-intestinal disturbance increases; the nausea becomes constant, vomiting occurs, and diarrhœa is added to the other causes of depression. Under these circumstances it may happen that death ensues before the occurrence of the characteristic pigmentation. No case, however, can be regarded as strictly typical in which the peculiar bronzing of the skin is entirely wanting. Those parts of the body exposed to the light first exhibit the change in color. The skin, in patches and streaks, and especially about old cicatrices, begins to grow dusky, or grayish brown; then assumes an olive-green tint, and becomes, finally, bronze or copper-colored. As the tint deepens, it also widens, until the whole surface is dark—mulatto-like—those parts naturally pigmented staining most deeply, while the palms and soles, the nails, and the sclerotic remain white. The mucous membrane of the mouth, also, exhibits patches of pigmentation about the lips and cheeks, but especially along the margin of the teeth. Nothing could be more striking than such a change in the color of the integument, but not all cases present it in full, and an early termination may prevent anything like a general deposit of pigment. There may be in such cases only patches of pigment here and there, in situations where the coloring matter normally abounds, at the site of old cicatrices, and at points of pressure of clothing. Although cases undoubtedly exist in which all the symptoms of Addison's disease are present, save the pigmentation, they should not be regarded as examples of this affection, unless an early termination prevents the full development. It has been suggested that cases of this disease have indeed proved fatal before the peculiar pigmentation could develop, by a persistent and uncontrollable vomiting, coupled with the usual asthenia. Sometimes diarrhœa has been associated with the gastric derangement, and the progress of the case accelerated by the gastro-enteritis, to which the fatal result was attributed. The simultaneous occurrence of so much pain and soreness in the epigastrium, of vomiting, and of extreme weakness, seems so much like the history of abdominal cancer, that a fatal result occurring before the pigmentation is attributed to cancer.

Course, Duration, and Termination.—The typical examples of Addison's disease are characterized by slowness of evolution. There are, however, cases much more acute in type, but these are decidedly in the minority. In these acute cases, after a period of unaccountable decline in health and activity, the patient goes to bed intensely prostrated, can hardly raise himself up, and on any muscular movements the members tremble. Nausea, vomiting, meteoric distention of the abdomen, and diarrhœa supervene, rapidly reducing the flesh and strength. The pulse grows quick and small, and the action of the heart, though rapid, is excessively weak. In these cases, usually, there is considerable febrile disturbance, quite in contrast to the subnormal temperature of the chronic type.

For many months in the ordinary cases the progress is very slow; apparently, indeed, unchanged. Meanwhile the asthenia increases, the gastric disturbance grows worse, frequent vomiting occurs, and a severe, sometimes uncontrollable, watery diarrhœa sets in. Fainting on attempting to rise, severe *tinnitus aurium*, headache, failure of memory, great mental feebleness, muscular twitching, epileptoid seizures, are in turn experienced as the case progresses toward the end. Various abnormal nervous manifestations occur, and in one case (Broadbent's) choreic attacks came on. Meanwhile, the abnormal pigmentation increases until the whole body becomes intensely bronzed. The only termination is by death. In a large proportion the immediate cause of death is phthisis—tuberculosis frequently coexisting with the disease, if, indeed, Addison's disease is anything else than tuberculosis of the supra-renal bodies. The average duration of the cases is one and a half year. The most acute cases last about six months, while the most chronic extend over several years (Wilks). The duration is affected chiefly by the progress made in the general tuberculosis, or in the phthisis, associated with the majority of cases. Rossbach treats of the association of Addison's disease with scleroderma.* In many instances caries of the vertebra, psoas abscess, and inflammation and suppuration in the neighborhood of the capsules have existed.† The progress and termination may therefore be affected by these associated lesions. Those cases in which the usual coexistent diseases are wanting, and the anatomical changes are restricted to the supra-renal capsules, terminate by asthenia, and are the most protracted.

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* Virchow's "Archiv," Band li, p. 100.

† Greenhow, *supra*.

Diagnosis.—Until the characteristic discoloration of the skin appears, the diagnosis must be largely conjectural. A persistent and unaccountable asthenia, coinciding with an increase in the depth of color of the natural pigment, and the appearance of grayish-brown spots, ought to be rightly interpreted. Brownish spots appear on the face and other parts in some women during pregnancy and at each menstrual period, but in these cases the peculiar asthenia is absent. Greenhow describes * a case of "Vagabond's discoloration," which simulated Addison's disease; but the diagnosis was rendered easy by the free application of soap-and-water. Abnormal pigmentation occurs in some cases of exophthalmic goitre, but, while in this disease there is a marked degree of asthenia, the other symptoms of the morbid complexus readily determine its character. Anæmia should not be confounded with asthenia. The anæmia of chronic malarial poisoning, accompanied by more or less fawn-color of the skin, may be mistaken for the asthenia of Addison's disease. The distinction ought to be made between anæmia and asthenia, which will be confirmed by the previous history in both diseases. The discoloration of the skin when general may be confounded with jaundice, but the distinction is made by the extreme weakness which accompanies Addison's disease, and by the fact that in jaundice the change of color extends to the eye, to the mucous membrane, and to pathological fluids as well as to normal excretions, as the urine. Pityriasis versicolor is readily distinguished by the roughness of the skin, by the occurrence of the discoloration in patches, and by the presence of a parasitic organism. Furthermore, pityriasis versicolor does not materially affect the general health, and is not accompanied by the profound depression characteristic of Addison's disease. Leucoderma is readily diagnosticated by the accompanying patches of pigmentation and abnormal whiteness of the skin.

Treatment.—The treatment, although unpromising, is not without utility. There are two objects to be attained by remedies: to improve the cachexia; to relieve the more active symptoms. In the first group are such remedies as the sirup of the iodides of iron and manganese, cod-liver oil, chloride of calcium, quinia, iron, etc. Phosphorus has been given with obvious good results in several instances, and the phosphates and phosphites are excellent reconstituent tonics, which may be useful in this disease. Arsenic, also, is a promising remedy. Probably the best effects will be obtained from the administration of phosphorus in cod-liver oil, and the chloride of calcium with the sirup of the iodides.

For the nausea and vomiting, minute doses of Fowler's solution, bismuth and carbolic acid, and hydrocyanic acid, may be in turn tried. Tincture of nux vomica is an excellent stomachic tonic under these circumstances.

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* "The Clinical Society's Transactions," vol. ix, p. 44.

MELANÆMIA.

Pathogeny.—The term *melanæmia* is applied to a condition of the blood in which are found small brownish or black masses, scarcely so large as a red-blood globule, of pigment matter. Sometimes these particles are oval, or round in shape, sometimes irregular, and rarely stratified by the presence of a colorless capsule (Rindfleisch). Occasionally true pigment-cells are observed. This pigment is found everywhere in the blood, but exists in greatest quantity in the spleen, which becomes, according to the quantity, a chocolate, brownish, or blackish color. The spleen may, indeed, be almost the sole place of deposit, but the liver is next in respect to place and quantity, and after the liver are the lungs, brain, and kidneys. Opinions differ as to the origin of the pigment, but the weight of authority is in favor of the splenic origin, and that it is a product of the disintegration of the red-blood corpuscles. As during malarial fever this destruction of the red corpuscles is more rapid than in any other form of acute infectious disease, melanæmia is a product of malarial diseases. The pathological changes characteristic of this state are found in the spleen, liver, lymphatic glands, marrow of bones, etc. The spleen is enlarged, its consistence soft, if there have been recent attacks, and firmer if considerable time has elapsed. The color depends on the quantity of pigment, and is dark slate, or brown, or black. The deposits of pigment take place chiefly along the veins, which are bordered by a dark line, and to a less extent along the arteries, and the whole splenic pulp may be tinted by it. The lymphatics and the marrow, also, contain pigment, which, with lymphoid cells, is found in the vicinity of the vessels. Characteristic changes, due to pigment deposition, also occur in the liver. As elsewhere, the pigment deposits are found alongside the vessels. According to Rindfleisch,* small extravasations of blood in Glisson's capsule, and in the parenchyma of the liver, initiate the pigment formation. The pigment granules accumulate about the branches of the portal vein and hepatic artery, about the intralobular and hepatic veins, but the hepatic cells are not involved. The whole organ has a steel-gray or blackish tint. Ultimately the nutrition of the organ may be so impaired that atrophy results.

As the pigment granules may be larger in caliber than the blood corpuscles, they will necessarily be arrested in those organs having a fine capillary network. Pigment embolisms of the cerebral vessels are, consequently, results of this process. Pigment blocking of the cerebral capillaries has precisely the same effects that other emboli produce: collateral hyperæmia, extravasations, and œdema, with the important structural alterations following in their wake.

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* "Pathological Histology," American edition, p. 187.

Symptoms.—Melanæmia is an accident or complication of the severer cases of malarial fever. The changes in the spleen and liver do not cause symptoms, except the enlargement of the former organ, to be made out by palpation and percussion. The cerebral symptoms are, however, very pronounced. There are present, when the pigment embolisms occur, more or less intense headache, vertigo, delirium either low-muttering, or active and furious, passing into stupor, coma, and insensibility. There are occasionally paralysis and epileptiform attacks, but usually the motor disturbances are not more than twitchings of the muscles, ptosis and weakness of the muscles of the extremities. In cases seen by the author the delirium was wild—delirium ferox—and the motor troubles were those of paresis of muscular groups. In the author's cases also there was a very high temperature, to which the cerebral disturbance may have been in part due. In the more chronic cases, without fever, there are persistent headache and vertigo, the strength is easily exhausted, the nutrition inactive, and the surface, especially of those parts of the body exposed to the light, has a bronzed appearance. In such, we may assume that the pigmentation of the brain is confined to deposits alongside the vessels, and does not include embolic obstruction of the capillaries by pigment masses. When the last-mentioned condition exists, there will be more decided mental symptoms, epileptiform attacks, paralysis, etc. In the milder form, recovery may ultimately ensue if the patient be removed from miasmatic influences. In those cases of capillary embolisms, it is doubtful if recovery ever can take place. Nevertheless, treatment must be pursued from the symptomatic standpoint, for it may be that success will eventually be the reward of persistent efforts.

Treatment.—There are two therapeutical indications: to check the waste of red-blood globules; to effect the solution and extrusion of pigment. Quinine, iron, ergotin, and digitalis—which may be combined—are the most efficient remedies for the first indication; pyrophosphate of sodium for the second. If the symptoms are acute, quinia must be given in large doses—twenty to forty grains a day—if less so, five, even three grains three times a day. The other remedies should be prescribed accordingly.* The utility of the phosphate of sodium consists in its power to maintain the alkalinity of the blood, in its effects on the hepatic secretion, and in its influence over the metamorphosis of tissue.

HÆMOPHILIA.

Definition.—The term *hæmophilia* is applied to a congenital state characterized by the habitual occurrence of hæmorrhages. As the disposition to bleeding is inherited, and is transmitted in families, persons so affected are called "bleeders."

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* ℞. Quiniæ sulph. ʒ j, ferri redacti gr. x, ergotin ℈j, digitalis gr. x. Make into ten wafers. One wafer three times a day. ℞. Sodii pyrophosphat. ℥ j, ferri pyrophosphat. ℈j. M. Take a teaspoonful in sufficient water three times a day before meals.

Causes.—Heredity is the most important factor in its causation. It is an unfortunate fact that families of bleeders are remarkable for fertility. The males are affected thirteen times more frequently than females (Immermann *), but, on the other hand, women transmit the disease more certainly than males—for example, a male bleeder marrying a healthy woman, without taint of hæmophilia, has children usually free from this hereditary disposition; but a female bleeder marrying a healthy male has quite uniformly bleeder children. Again, if a woman, member of a bleeder family, but herself not a bleeder, marry, she will have some children who inherit the family taint. The disposition to bleeding usually manifests itself about the first dentition, and in a large proportion within the first year. The hæmorrhagic diathesis existing, a slight injury will suffice to start the bleeding: thus, lancing the gums, leech-bites, the Jewish rite of circumcision, slight cuts or abrasion of the skin, have been followed by uncontrollable hæmorrhage. The bleeding having once occurred, the tendency to attacks is thereby greatly increased.

Symptoms.—There does not seem to be anything peculiar in the bleeders as respects bodily conformation, temperament, habits, and disposition, except the hæmorrhagic diathesis, although it is said that they are usually persons of superior mental endowments (Legg †).

There are two distinct forms of hæmorrhage: the *external*, in which the blood pours out on the surface of the wound or abrasion; the *interstitial*, in which the blood diffuses into the interstices of the adjacent tissues. Frequently, if not usually, both forms occur at the same time. The external form may be the result of injury, and is therefore *traumatic*, or it occurs *spontaneously*, and is named accordingly. The external and traumatic form is single, for it is comparatively rare for more than one point of injury to exist at a time. On the other hand, the spontaneous hæmorrhage, indicating a more active state of the vice, may occur simultaneously at several points. The most usual site of the spontaneous hæmorrhage is the mucous membrane, especially of the oral and nasal cavity; of the stomach and intestines; of the bronchi; of the genito-urinary passages—named in the relative order of frequency. Recent cicatrices, that are still vascular, ulcers of the skin, and irritated surfaces, invite the hæmorrhage. Again, in the most perfect specimens of hæmophilia, bleeding occurs without any change in the skin to start it, and takes place from the fingers, toes, lobes of the ears, back of the hand, etc. By far the most common form of bleeding is epistaxis, which occurs, according to the statistics of Grandidier,* four times more often than hemorrhage from the gums, which comes next in frequency, then intestinal hæmorrhage, hæmoptysis, hæmaturia, hæmatemesis, etc., as named.

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* Ziemssen's "Cyclopædia," vol. xvii, article "Hæmophilia."

† Dr. J. Wickham Legg, "Treatise on Hæmophilia," London, 1872, H. K. Lewis, p. 158.

The blood escapes from the smallest capillaries, under very strong pressure, and persists obstinately, in spite of the most powerful means to arrest it, hours, days, and weeks together. The result is an extreme degree of anæmia—the skin pallid, the face drawn, lips retracted, the mucous membrane white and sticky, the pulse small, weak, or not to be felt at the wrist; a soft, systolic murmur at the base, and a venous hum over the great veins; or the action of the heart may be too feeble to be recognized. Consciousness may be lost, and death occur in syncope. Owing to the extreme cerebral anæmia, there may be illusions, hallucinations, or attacks of convulsions, as in animals bled to death (Kussmaul and Tenner †). In the syncope, a hemorrhage which could not be arrested may cease spontaneously. Notwithstanding the enormous losses of blood, its reproduction takes place quickly, and between the seizures the bleeders may present the rosy hue of health. The amount sometimes lost seems almost incredible—in one case (Coates) reaching the enormous loss of three gallons in eleven days. The state of the blood in bleeders varies with the conditions of health and after loss by hæmorrhage—that is, becomes more watery with loss—but otherwise there is no difference in composition as compared with healthy blood, except that the former contains somewhat more red globules and more fibrin than the latter, or is richer than ordinary normal blood. The interstitial bleedings occur chiefly in the skin and subcutaneous connective tissue, and when traumatic are observed in parts subject to injury, as the back, buttocks, trochanters, while the spontaneous are observed mostly on the scalp, the scrotum, and the legs. Very small extravasations are called *petechiæ;* larger ones, *ecchymoses.* The blood undergoes the usual changes of extravasated blood: at first a bluish red, then brownish, with green borders, then yellowish—several weeks being occupied in these transformations. Sometimes considerable accumulations of blood are formed, constituting blood-tumors, and are found about the false ribs, on the back, on the inner face of the thighs, in the popliteal space, and on the lower extremities. They vary in size from a hickory-nut to a goose-egg, and attain even larger proportions, and also vary in firmness according to their position. They are of a bluish-black color, and are surrounded by a rose-colored zone, tender to the touch, and signifying the formation of a limiting membrane. These tumors may undergo the usual preparatory changes and be slowly absorbed, or suppuration may occur, and discharge of pus and shreds of tissue take place, with considerable hæmorrhage. The only changes to account for the phenomena of hæmophilia are abnormal disposition and arrangement of the superficial vessels of the body. The superficial vessels are abnormally large, the intima remarkably thin. On the other hand, the lumen of the large arteries (aorta and pulmonary) is found to be narrow. The intima of both classes of vessels is usually in a state of fatty degeneration. There has usually existed an hypertrophy of the left ventricle. These changes in the vascular system, and the condition of vascular fullness and congestion, which marks the healthy state of bleeders, together with the abnormal richness of the blood, serve in a measure to account for the extraordinary clinical history of this disease.

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* Schmidt's "Jahrbücher," vol. cxvii, p. 329, "Bericht über die neuern Beobachtungen und Leistungen ein Gebiete der Hæmophilie seit, 1854."

† Sydenham Society edition.

Complications.—In the bleeder families neuralgic and rheumatic affections seem common. Toothache and myalgia are said to be frequent. Rheumatic joint and muscular affections also occur.

Duration and Termination.—The duration of hæmophilia is the life of the individual. If the bleeder escape the accidents of childhood, there may be no manifestation of the diathesis until after adult life. A young woman died on her marriage-night, from hæmorrhage occasioned by rupture of the hymen. A single hæmorrhage may take life in a few hours, as in the case just narrated, or death may result from several weeks of bleeding. The usual result is death. Such small operations as extraction of teeth, circumcision, leeching, etc., are very apt to cause death, while vaccination is much less dangerous. Of 152 bleeder boys, 133 died before attaining twenty-one years of age.* The hæmorrhagic disposition may disappear in middle life, but this has happened in nine cases only; and, when it does cease, rheumatic and gouty attacks are experienced.

Treatment.—All injuries must be carefully guarded against. Bleeding from any abrasion or puncture should be restrained by pressure, if possible. Every form of astringent vegetable and mineral has been used. Epistaxis, which is the most usual form of hæmorrhage, is best arrested by plugging the nares and the application of ice, and by the administration of ergotin. Bleeding from the gums is more easily handled, in that the styptic preparations of iron, the actual cautery, and compression can be used. In hæmaturia, krameria, infusion of digitalis, ergotin, and gallic acid should be administered. Of the systemic remedies there can be no question as to the superiority of ergot and digitalis, and experience is in harmony with physiological experiment. Cures have apparently followed the use of ergot. The administration should never be subcutaneously, and the dose of the aqueous extract will range from two to five grains, as often as may be necessary. When attacks are impending, a brisk cathartic of Epsom salts should be administered to lower the blood-pressure, and the diet should consist of fruits and vegetables only. Sulphuric acid in dilute solution should be taken as a drink. Full doses of digitalis, the patient maintaining absolute recumbency, should then be administered, and when the hæmorrhage comes on the exhibition of ergotin, etc., should be practiced. This method is the best now known for arresting the attacks of bleeding.

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* Grandidier, *op. cit.*, p. 333.

SCORBUTUS—SCURVY.

Definition.—Scurvy is a disease of nutrition, in which the blood is so far impoverished that transudations occur, and large hæmorrhagic ecchymoses become visible in various places.

Causes.—This disease occurs more frequently in men, because their occupations expose them more to its causes, and in the feeble and cachectic, especially those who are debilitated by syphilis and mercurialism, and by marsh-miasm. Scurvy usually happens in bodies of men, as soldiers and sailors, who are under the same evil influences, and hence numbers are attacked nearly simultaneously—the cachectic falling victims before the robust. The chief factor is defective alimentation, not in respect to quantity so much as quality. The continued use of salted meat and fish and the absence of fresh meat and fresh vegetables for a long period from the diet are the great cause, and all other influences are merely adjuncts. When such fresh vegetables as potatoes, cabbage, and onions, are supplied, although the other components of the ration may consist of salted and dried meats, scurvy will not occur. So well is this fact understood now, that some one of these articles always enters into the diet of armies and prisons, and, if not attainable in a perfectly fresh state, are supplied in the form of "desiccated vegetables," *sauerkraut*, etc. Garrod, and afterward Hammond, attempted to show that the constituent, the absence of which is the cause of scurvy, is potash; and that those vegetables most effective in preventing and curing scurvy are remarkable for the quantity of potash which they contain, and of these the potato stands at the head. Undoubtedly, bad hygienic influences exert an influence in the production of scurvy. Living in houses that are dark, damp, and confined, want of exercise, depression of spirits (defeat), *ennui*, all have more or less effect in depressing the bodily functions, and thus favor the ill effects of an improper diet.

Pathological Anatomy.—Cadaveric rigidity is slight; suggillations are extensive on the dependent parts; petechiæ and ecchymoses are found on the body and the extremities; the skin is muddy, inelastic, and scaly. The petechial spots are formed by an extravasation proceeding from the capillary network about the hair-follicles, while the larger ecchymoses come from the vessels of the derma. The indurations of the connective tissue, subcutaneous and deeper, are due to infiltration by coagulated blood. The subsequent changes in the clots are the explanation of the appearance presented by these indurations, and depend on the greater or less amount of red globules, and on the solution of the fibrin, or its organization. The fibrin may become organized to that extent in which muscular atrophy and contractions resulting in deformities must ensue. In a similar manner, an extravasation into the substance of a muscle may lead to atrophy, the muscular elements being supplanted by indurated connective tissue. These atrophic alterations and deformities are results of long-standing changes. Recent extravasations, in scorbutus, under appropriate management, undergo the same regressive changes as a blood-clot in the normal state, though somewhat slower, and nothing is found *post mortem* after the process is completed. The mucous membrane of the mouth is the seat of extensive hæmorrhagic infiltration, and is therefore swollen and spongy ; but in old cases the gums may be thickened and indurated, due to the formation of new connective tissue. There is more or less effusion into the serous cavities of a straw-colored or sanguinolent serum ; the membranes are injected, or coated with exudations, or stained by spots of hæmorrhagic extravasation. The heart is flabby, soft, pale, and hæmorrhages are found in its muscular substance. The lungs are somewhat œdematous, the posterior and dependent parts the seat of hypostatic alterations, and catarrhal and croupous inflammation products are found at the base and elsewhere. There may be extensive solidification from croupous pneumonia, or hæmorrhagic infarctions. There are numerous ecchymoses in the bronchi. The peritoneum is altered in the same manner as the pleura—the evidences of inflammation existing on the visceral and parietal layers in the form of exudations and extravasations. The intestinal mucous membrane is altered by hæmorrhagic spots and erosions, and sometimes by extensive losses of substance. The liver is not usually affected. The spleen, although often unchanged, is sometimes enlarged and softer than normal, and occasionally there are found hæmorrhagic infarctions. The kidneys may be healthy, but the mucous membrane of the pelves, ureters, and bladder contains erosions and ecchymoses. Important alterations occur in the blood—the number of red globules diminished ; the white relatively increased ; the iron, potassa, and albumen lessened.

Symptoms.—The onset of scurvy is so gradual that the patients do not know when it began. They become a little paler, and fatigue more readily, but after a time there is an appearance of anæmia, and such a degree of weakness that the least effort gives rise to exhaustion, and to a sense of præcordial oppression and weakness and palpitation of the heart. The increasing weakness is accompanied by a sense of soreness and fatigue in the muscles, like that induced by prolonged hard work, but rest in bed relieves, as exercise increases, these sensations. These muscular pains are especially felt in the back and the calves of the legs, and have a rheumatic character, and are often supposed to be rheumatic. The scorbutic subjects become exceedingly sensitive to cold, and continually seek the fire or put on additional clothing. They are somnolent, apathetic, and indisposed to any effort, mental or physical; are dejected in mind, and wear an expression of sadness. The facies presents an unearthly aspect; the eyes are sunken and surrounded by livid aureola; the lips are thin, retracted, cyanosed; the skin sallow, pallid, dry, scaly, and earthy, and here and there may be found indistinct spots of bronze discoloration. The subcutaneous fat has diminished, the muscles are soft and small, and the body-weight is reduced. Such are the symptoms of the initial or prodromal stage. They indicate anæmia, and are suggestive of scorbutus only because of the surroundings, and the presence of other cases. The duration of this period is from a week to two or three months. This prodromal stage may be wanting, but in the cases observed by the author * was always present.

The scorbutic stage first manifests itself in the gums, which become of a dark-bluish color on their margins, especially at the incisor teeth, and are swollen, projecting between the teeth, and bleeding with a touch. The gums are also quite painful, so that mastication and the mere contact of sapid substances are distressing; but those portions of the gums without teeth are free from these troubles, and hence the toothless, at the extremes of life, are exempt from scorbutus of the mouth. Again, it sometimes happens that these changes in the gums are entirely absent, and the first manifestation of trouble consists in suggillations and subcutaneous extravasations of blood and intestinal hæmorrhage. On the other hand, there are many instances in every collection of cases, in which the only manifestation has been in the mouth, coupled with anæmia and muscular feebleness. In the severer cases after the prodromal stage, the weakness increases to such an extent that they become unable to retain the upright posture, and will fall into syncope in the attempt to assume this position. The action of the heart becomes very feeble, and any exertion brings on severe palpitation, with a sense of extreme præcordial oppression. Fever now comes on, in many cases not as a necessary element in the disease, but a symptomatic expression of a local inflammation of a serous membrane or other inflammatory trouble. The characteristic *bruit* of anæmia is audible at the base of the heart and along the great vessels.

In the further progress of the case the gums become much swollen, rise up to a level with the teeth, are horribly painful, and undergo ultimately an "ichorous disintegration," or diphtheritic sloughs form; in either case, fetid, decomposing sloughs are cast off, leaving the teeth bare or loose. Serious deformities are necessarily produced by these losses of substance when cicatrization occurs. Extensive hæmorrhagic extravasations take place in the skin, chiefly of the lower extremities and body, but rarely on the head or face. There may be purpuric petechiæ, the size of a hemp-seed, or vesicular or papular efflorescences, or large hæmorrhagic spots of irregular size, or vesicles of large size filled with a bloody serum. The least injury or contusion is followed by a suggillation. The skin, too, may become the seat of extensive ulcerations, gangrenous sloughs and hæmorrhage. The subcutaneous tissue may either suddenly or gradually become affected by indurations often of great extent. They are at first red, and tender, but presently become brownish, and the epidermis peels off, leaving a discoloration; or, in severer cases, an acute inflammation is set up, the skin gives way, and a great quantity of blood with shreds of tissue, often gangreneous, is discharged, leaving a more or less extensive foul ulcer. The muscles undergo similar changes—are occupied by indurations, the result of extravasation of blood into their substance, and either acutely inflame, there being great local tenderness and heat, and symptomatic fever, or the process goes on more slowly without fever. Hæmorrhages take place from various mucous surfaces: epistaxis; hæmatemesis; intestinal hæmorrhage; hæmaturia. Fortunately, hæmorrhage from the broncho-pulmonary mucous membrane is not common, except in cases of incipient phthisis. Hæmorrhages take place also on the serous surfaces, and hæmorrhagic effusions, the result of inflammation, are not infrequent in the pleura, pericardium, and peritoneum. Enlargement of the spleen, often to a considerable extent, occurs in a portion of the cases. Albuminuria is present in the severer cases very often, and the urine is otherwise changed in character and composition. The most notable change besides the albuminuria, is the diminution, not only in the amount of urine secreted, but in the relative amount of its solids.

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* The author saw some cases of scurvy when serving in the regular army as medical officer in 1857, during the winter spent in Utah, the command being on half rations, without any fresh vegetables. The description above is, in the main, based on these observations.

Complications.—The periosteum, cartilages, and joints are affected in the worst cases. Extravasations take place under the periosteum, causing a painful swelling, which may take on an inflammatory character if the extravasation be large. The epiphyses of the long bones become swollen, soften somewhat, and may be detached even. Hæmorrhagic effusions occur in the articulations, causing painful swelling, inflammation, and fever. Meningeal hæmorrhage is a very rare accident, but hæmorrhage into the substance of the brain never occurs. Extravasations of blood also take place in the anterior chamber of the eye and under the conjunctiva. Severe inflammation may be the result. Hemeralopia, or night-blindness, has long been associated with scurvy, but cases of scurvy are without it, and it often exists quite apart from scurvy. The profound alteration in the fluids and solids of the body caused by scorbutus invites attacks of other maladies. A frequent complication is croupous pneumonia, and a cause of death in many cases. Hæmorrhagic infarctions, usually several, sometimes are also found in the lungs. Ulcerative endocarditis and hæmorrhagic pericarditis are complications which quickly cause a fatal result.

Diagnosis.—Until the characteristic change has occurred in the gums, on the skin, etc., the anæmia of scorbutus is not distinguishable from other diseases characterized by this state. When, however, the gums swell, and there are petechiæ on the skin, and indurations beneath, it is impossible to confound it with any other malady.

Course, Duration, and Termination.—The usual course of scorbutus consists in the prodromal period, the fully developed attack characterized by the swollen and sloughing gums, the hæmorrhagic affections of the skin, the extravasations into the subcutaneous areolar tissue and muscles, the inflammatory hæmorrhagic exudations of the serous membranes, the profound cachexia, and the period of restoration. The duration is usually protracted, and is influenced by the hygienic surroundings. When the disease is fully developed, the continuance of the causes will keep it in action and increase the morbid process, while recovery, even in an apparently hopeless condition, takes place promptly when the proper aliment is supplied. The earlier the appropriate means of cure are applied, the more perfect the restoration. Serious deformities may result from the inflammations of the muscles, bones and joints, and death quickly follows the lighting up of pleuritis, endocarditis, peritonitis, etc. These evil results only occur when the disease has been unusually protracted and severe. Death usually results from hæmorrhages, from exhaustion, from a serous inflammation, or from pneumonia, but the mortality depends almost wholly on the failure of the necessary supplies, and not on the virulence of the disease. With the progress of knowledge, scorbutus is becoming much less common. No longer are witnessed the frightful cases in armies, on shipboard, and in prisons, such as were very common only a century ago.

Treatment. — The prophylaxis as well as treatment of scurvy, above all things, necessitates the use of anti-scorbutic food, fresh vegetables of all kinds, especially the potato and sauerkraut, and lime-juice. In the English navy, lime-juice is most depended on; but ships and bodies of troops are also supplied with "desiccated vegetables," the ordinary vegetables, including cabbage, onions, potatoes, etc., compressed into tablets and carefully dried. Desiccated or condensed milk is also utilized for the same purpose. Whenever attainable, fresh meats are extremely serviceable, and, in their absence, canned meats, beef-juice, and similar preparations, can be made to supply their place. Yeast has been found by Neumann * to be highly beneficial, and also the barm of beer. Medicines play a secondary part in the treatment of scurvy. In accordance with Garrod's and Hammond's potassa theory, we may prescribe cream-of-tartar lemonade, to be drunk freely. Quinine and sulphuric acid, either alone or in combination, are used to diminish transudations and to improve the tone of the system in general. Tincture of the chloride of iron and ergot are given to arrest hæmorrhage. There can be no doubt, if the author can depend on his own observation, of the value of whisky as a remedy for the scorbutic state, and to lessen or prevent the extravasations of blood. An ounce of whisky every four hours is generally the most useful amount. Turpentine is a highly efficient stimulant and hæmostatic under the same conditions, and is the best dressing for the ulcers in the skin. Alum, tannin, subsulphate of iron, and chloride of iron, are the most useful local styptic applications for arresting epistaxis, and hæmorrhage from superficial wounds, or ulcers of the skin. Ergotin can, at the same time, be administered by the stomach. Red cinchona-bark in powder is an excellent dressing for the ulcers of the skin. As the various manifestations and localizations of the disease are due to the cachexia, no time should be wasted in treating them, but every effort put forth to improve the condition of the body in general.

PURPURA—PURPURA HÆMORRHAGICA—MORBUS MACULOSUS.

Definition.—The term *purpura* means a bluish-red or purplish discoloration, produced by extravasation of blood; *purpura simplex* is applied to the simplest form of this malady, in which there are only minute extravasations in the skin (petechiæ), and no hæmorrhages into other parts; *purpura hæmorrhagica* indicates a condition of things in which not only petechiæ appear in the skin, but ecchymoses, vibices, and hæmorrhages occur. Besides the variations in intensity as expressed in the names applied to the disease, there are differences in character. Although a very large proportion of cases of purpura, whether simple or hæmorrhagic, are entirely free from fever, there are cases of both forms in which fever is present—the febrile form (purpura febrilis). There are other cases, complicated with rheumatism, one or several joints being affected—rheumatic purpura (purpura rheumatica).

Causes and Symptoms.—Purpura is not limited by climate, race, sex, or social condition, but it occurs more frequently in females, and is more common from fifteen to twenty than at any other age. It appears to be strictly sporadic. Convalescents from fever seem to be specially liable to it. The disease usually begins abruptly, the first manifestation being epistaxis. In a few cases there is a prodromal period, of a few days, possibly a week, in which there are some languor and inaptitude for exertion of any kind, sometimes with feverishness, sometimes with rheumatic pains, and slight swelling of the joints, usually the ankles and knees. The next symptom is the occurrence of petechiæ on the lower extremities and body, less on the arms, and rarely on the face. These petechiæ or bluish-red spots, vary in size from a pin's-head to a pea, and change in color successively from bluish-red to greenish, brown, and yellow. As successive crops come out, the appearance of the skin is peculiar, the different colors of different ages being curiously intermingled. Slight injuries, blows and contusions, are followed by extravasations, bluish-red spots of irregular size making their appearance. So long as the disease is limited to these manifestations, it is entitled purpura simplex; but hæmorrhage from the mucous surfaces is very common. The mucous membrane of the mouth is a not unusual source of hæmorrhage, but the spongy and sloughing gums of scurvy are entirely wanting, as also the diphtheritic and inflammatory exudations. Hæmorrhages may also occur in the subcutaneous areolar tissue, in the serous cavities, from the cerebral meninges, but these are exceptional; whereas the hæmorrhages from the mucous surfaces is the special feature, and may be the only condition present. It has been observed a few times that the hæmorrhages have come on suddenly, without any other symptoms, in apparently healthy and vigorous subjects, and without impairing the general health; usually, however, the repeated losses of blood cause an extreme degree of anæmia, manifested by pallor, emaciation, weakness and breathlessness on slight exertion, faintness on assuming the erect posture, swollen ankles, etc. Before hæmorrhages occur, the condition of the blood seems normal; but in the further progress of the cases the blood becomes watery, the white corpuscles increase in number relatively, and the red corpuscles decrease, but the coagulability of the blood is at no period lost. Besides the presence of blood on the mucous surfaces and on some of the serous membranes, there are *post-mortem* changes to be noted. The hæmorrhages are mere extravasations, and under no circumstances inflammatory. The disease may therefore be regarded as a "*transitory hæmorrhagic diathesis*" (Immermann). An important result of the disease, due directly to the hæmorrhages, but persisting after they have ceased, is anæmia. It is in a high degree probable that the anæmia, which is increased by the hæmorrhage, is also a principal factor in their causation. Urticaria is another complication, and seems to be associated with stomach derangement. A much more rare accident is the occurrence of sloughing and perforation of the intestines, produced by hæmorrhagic extravasations into the tunics of the bowel.

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* Immermann, *op. cit.*

Course, Duration, and Termination.—The whole course of the disease includes the prodromal period, the purpura simplex, the period of hæmorrhage, and the subsequent anæmia. The duration is influenced materially by the number and amount of the hæmorrhages. An ordinary case will last two or three weeks, but when there are repeated hæmorrhages the disease may continue for several months. Although most cases recover, death sometimes happens from exhaustion, from internal hæmorrhage, from some intercurrent malady, and from perforation of the bowel.

Diagnosis.—Purpura may be confounded with scorbutus, hæmophilia, progressive pernicious anæmia, leucocythemia, and cerebrospinal meningitis. From scurvy it is differentiated by the absence of changes in the gums, of the indurations of the subcutaneous areolar tissue and of the muscles, of the hæmorrhagic inflammation of the serous membranes, etc. From hæmophilia the distinction is made by reference to the history, especially the heredity, by the period of life, by the bleeding from trivial wounds, so characteristic of hæmophilia, and not of purpura. The distinction of purpura from progressive pernicious anæmia rests on the fact that in the former the anæmia is produced by the bleeding, in the latter the bleeding comes on afterward and is due to the poverty of blood. From leucocythemia the distinction is made by the enlarged spleen and enlarged lymphatics, with the growth of which a marked degree of anæmia is coincident, and to which the hæmorrhagic tendency succeeds. The initial symptoms of cerebro-spinal meningitis may be almost identical with those of purpura : purplish spots, pains in the joints, with some slight feverishness, but in a day or two the occurrence of nervous phenomena decides the question.

Prognosis.—Most of the cases terminate in recovery. A guarded opinion must be expressed when the hæmorrhages recur again and again, and when the disease occurs in broken-down subjects.

Treatment.—The usual treatment consists in the administration of the mineral acids, especially the sulphuric, and of the preparations of iron, especially the tincture of the chloride. With these remedies must be conjoined a suitable dietary, fresh air, sunshine, and moderate exercise. If constipation be present, the most appropriate laxative is sulphate of magnesia with dilute sulphuric acid. If hæmorrhages that are threatening come on with a strong pulse, flushed face, headache, and excitement, digitalis, quinia, and ergotin are the appropriate medicaments. If there be weakness and debility, quinine and alcoholic stimulants moderately should be prescribed. The local means for arresting bleeding consist in subsulphate of iron, tannin, alcohol, ice, or it may be hot water, which is sometimes more effective than cold. For the after-anæmia iron should be pushed.

ANÆMIA—OLIGÆMIA.

Definition.—The term *anæmia*, which signifies want of blood, consists of a deficiency of its nutritive constituents. *Oligæmia*, which signifies poverty of blood, is a more correct term; but the former is too firmly fixed by usage to permit a change. Although from the etymological point of view anæmia must be used to indicate a deficiency of blood, yet, by common usage, it is understood to mean poverty of the blood, and in that sense is employed in this work.

Causes.—The tendency to anæmia is influenced by sex, age, and peculiarities of individual constitution. The female sex is more liable than the male, for the reason probably that the former are by nature less endowed with the nutritive constituents of blood. Compared to the body-weight, and still more decidedly by sex, the blood of women contains fewer red corpuscles, more water, and less albumen and salts, than the blood of men. While the average number of red globules in the blood of healthy adult males is 141·1 per 1,000 parts, in the healthy adult female it is 127·2 (Becquerel and Rodier*). The extremes of life—youth and old age—are more liable to anæmia than the period of maturity. In early life the needs of the growing organism are such as to require the utmost amount of pabulum from the blood; the interchanges are more rapid, the consumption of material greater, and hence the more ready development of anæmia if other circumstances coincide. In old age, on the other hand, the productivity is diminished, and hence the waste may easily exceed the demand if there be any disturbance either in the preparation of materials for the blood or in the retrograde metamorphosis of the tissues. There are those also who have a natural tendency to anæmia, a peculiar type of constitution. They are in a condition the opposite of plethora, are deficient in the amount and quality of blood, and seem to be unable to produce it effectively. Sometimes they are persons of full habit, but possess a lax fiber, and are pale and weak.

A powerful exciting cause of anæmia is an insufficient supply of food. Again, the food being abundant, anæmia may be the result of poor digestion, and faulty and imperfect assimilation. The food abundant, and the primary assimilation active, anæmia may result because of a deficiency in the supply of oxygen to complete the cycle of processes terminating in healthy blood. When the products of digestion are pouring into the blood, oxygen is needed to burn off the effete, excessive, or improper materials, and to perfect the preparation of the new materials. Light is also necessary to this process. Moderate exercise, by increasing the rate of organic movements and the consumption of oxygen, favors the preparation of the blood and improves its quality. The absence or imperfect supply of food, light, air, and exercise, impairs the vital processes and induces anæmia. Excessive exertion and fatigue, by the over-consumption of material, directly contribute to the production of the anæmic state. Heat acts similarly, in that prolonged high temperature increases the rate of circulation and the interchanges of waste and repair, while at the same time it interferes with supply by lessening the appetite and the digestion. Frequent repetition of the sexual orgasm, profuse menstrual flow, prolonged lactation, hæmorrhages, are very powerful causes of anæmia. Diseases of the organs concerned in nutrition, notably the digestive organs, malignant growths, albuminuria, the slow absorption of various mineral, vegetable, and gaseous poisons, and numerous pathological processes, either produce or are accompanied by anæmia; but in this relation the position of anæmia is quite secondary.

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* "Pathological Chemistry," translated by Dr. S. T. Speer. London: Churchhill, 1857.

Pathological Anatomy.—The changes found *post mortem* in anæmia from hæmorrhage are simply the appearances due to an exsanguine condition of all the organs and tissues. They are paler, drier, more compact, and free from blood. If death has been preceded by a wasting malady, not only is there the condition of bloodlessness, but the body is shrunken, the subcutaneous fat has disappeared, the muscles are thin, and the serous cavities contain more or less fluid. Patches of fatty degeneration occur in the muscular tissue of the heart—chiefly in the papillary muscles—and to the eye present the appearance of yellow spots and striæ. A similar (i. e., fatty) change is to be found in the intima of the great vessels, notably the aorta. Fatty change also takes place in the gland epithelium of various organs—the kidney epithelium, the hepatic cells, the gastric-gland epithelia, etc. The blood has a brighter tint than in the normal condition, due to a diminution in the number of red-blood globules, and in the quantity of hæmoglobin. In the anæmia due to loss of blood, the amount remaining after death is much below the normal; under other circumstances, the diminution may be but slight. The blood is also thinner, and has less power of coagulation, the clot lacking in firmness, whence it must be concluded that the fibrino-plastic substance and the fibrinogen are below normal.

Symptoms.—The simplest and purest form of anæmia is that caused by sudden and considerable loss of blood, as from wounds of arteries, unavoidable and *post-partum* hæmorrhage, etc. The symptoms are eminently characteristic: the skin becomes waxy white; the sclerotic pearly and glistening, eyes sunken; the face ghastly and shrunken; the lips pallid and bluish and retracted over the teeth; the nose pointed and cold; the finger-tips white, waxy, and cold; the surface of the body is cold, and the temperature reduced below the normal; the pulse is small, very quick, exceedingly feeble, and may cease to be felt at the wrist; actual fainting may occur; consciousness restored, fainting may be repeated, and this may occur many times; the attacks of syncope may be accompanied by epileptiform convulsions as in animals bled to death (Kussmaul and Tenner*); death may ensue in the syncope, or there may be a gradual restoration, the first change for the better consisting in a return of the pulse at the wrist, followed by warmth of the surface. But the weakness is yet extreme, and fainting occurs from the least exertion; or, when any effort is made, the face flushes, the heart beats rapidly, there is much oppression of the chest, and a sense of utter exhaustion. Excessive thirst is one of the immediate results of loss of blood, but the appetite for solid food returns very slowly. The urine is necessarily small in quantity after hæmorrhage, but the relative proportion of urea is increased. When restoration is taking place, the urea is less, the specific gravity of the urine falls below the average standard, until the normal state is reached. The most common form of anæmia is that induced by wasting discharges —prolonged lactation, for example—by disturbances in the function of nutrition—primary and secondary assimilation—by the cachexiæ—notably the malarial. This form of anæmia may be called *chronic*, while that already discussed is either acute or subacute. In chronic anæmia there exist pallor, or an earthy hue or fawn color of the skin, wasting to a greater or less extent, by disappearance of the subcutaneous fat, and a flabby state of the muscles: the skin is wrinkled, dry, and inelastic, the hair and nails appear dull and lusterless; the temperature of the surface below normal; the cutaneous circulation, the tension of the arteries, and the force of the cardiac contraction lowered; the anæmia *bruit* audible at the base of the heart and over the great venous trunks; sometimes a hæmorrhagic tendency develops; the function of digestion is wanting in energy, the appetite capricious, the bowels constipated; the urinary secretion is rather scanty, and may contain albumen, etc.; the sexual system is depressed, both male and female, and, while the sexual appetite is lessened in the male, amenorrhœa is present in the female, or there may be menorrhagia. Not all anæmic persons become paler by reason of diminished vascularity of the skin; those of dark complexion and the dark-skinned become darker. The emaciation, or at least the lessened fullness and roundness of the form due to anæmia, may be supplanted by œdema, produced by the changes in the composition of the blood. When the diminution of albumen reaches a certain point, the fluid normally contained in the tissue is not taken up by the blood-vessels, whence more or less œdema results, and, under the same circumstances, accumulation of serum takes place in the serous cavities. In this process there necessarily exist both "hypalbuminosis" and "hydræmia"—the former meaning a diminished amount of albumen; the latter, an increased amount of water. The hypalbuminosis is the most important factor in the production of the wasting or marasmus of anæmia. Not all parts lose in weight uniformly—the fatty tissue comes first, and next the spleen, liver, and voluntary muscles; and, as respects the muscular system, those waste least that are kept at work, as the heart and respiratory muscles. The weakness of the muscular system, which is so prominent a symptom in anæmia, is due largely to the diminished production of force, rather than to changes in the muscles themselves. The poor quality of the blood and the inactivity of the tissue-changes are the causes of the lessened evolution of force. A temperature below the normal is another result of the same causes. Among the most important of the symptomatic disturbances of anæmia are those of the nervous system. The organs of special sense are peculiarly alive to external impressions, and hence loud sounds, bright lights, and sharply sapid substances, make an unpleasant impression. The sensory and motor apparatus are similarly affected. Hyperæsthesia and hyperalgesia—neuralgia—are among the most disagreeable of the symptoms which occur during anæmia. Hysterical seizures, epileptoid attacks, are also results of an imperfect nutritive supply ("anæmia of the brain"). When the anæmia is extreme, as in cases of inanition, or from any cause, there is usually delirium, it may be, having a violent maniacal character, or low-muttering, or cheerful, busy delirium. The anæmia may result in syncope with temporary loss of consciousness—attacks frequently due to mere enfeeblement of the heart's action. As regards the condition of the organs of circulation, it is to be noted that the cardiac movements are feeble, the sounds muffled and indistinct, and the arterial tension low. The diminished power of the heart to move the blood leads to stasis in the venous system, which may result disastrously by œdema of the lungs, or hypostatic pneumonia, or by thromboses. More or less difficulty of breathing is a constant symptom, but there may be extreme dyspnœa when some sudden effort is made. The impaired breathing power is the product of several factors: 1. Of the increased irritability of the respiratory centers; 2. Of imperfect depuration of carbonic acid, and insufficient supply of oxygen.

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* "On the Nature and Origin of Epileptiform Convulsions, caused by Profuse Bleeding, etc." Sydenham Society translation.

Course, Duration, and Termination.—The course of anæmia is that of the malady with which it is associated or on which it is dependent. If due to hæmorrhage, or some sudden accident, it is acute, but the usual course is chronic. It has no defined duration, and is in no sense a self-limited disease. The progress of recovery is influenced by age, sex, and the recuperative powers of individuals. While women bear loss of blood better than men, they possess less restorative energy. The hygienic circumstances and the social condition are important elements in the process of reconstruction—for those who are most favorably placed have the best chance of recovery and the least delay in convalescence. Anæmia may result in death, in recovery, or in incomplete recovery. When the anæmia has been extreme, and the destruction of red-blood globules great, recovery is rarely, if ever, complete, and the patient's bodily vigor remains more or less below the normal.

Prognosis.—The cause of the malady and its associated states enter largely into the question of prognosis. When the anæmia is simple, due, for example, to sudden loss of blood, or to prolonged lactation, or to malarial infection, or to sexual disorders, or to diseases of digestion—all of which are perfectly remediable—the prognosis is favorable. When, however, anæmia has been produced by excessive loss of blood, and a condition of extreme debility has persisted for weeks; when associated with great mobility of the nervous system, and with protracted amenorrhœa, the prognosis must be guarded in respect to *complete* recovery. When anæmia is associated with cancer, albuminuria, suppuration of bone, amyloid degeneration, phthisis, scrofula, etc., the prognosis is unfavorable.

Treatment.—As the condition to be remedied consists in an impoverished state of the blood, obviously treatment must be directed to the organs concerned in the elaboration of blood; the organs of digestion, including the liver and pancreas, and the organs for the production of the corpuscular elements—the spleen and lymphatic system. The first step consists in the rectification of any existing disease of the digestive apparatus, if remediable; the second, in the supply of suitable aliment; the third, in the administration of certain medicines needed in the construction of the blood; and, fourth, in the admission of air, sunlight, and suitable exercise to an important place in the treatment, for these are required to perfect the final stage of the conversion of aliment into blood. If the digestion is feeble by reason of a deficiency of gastric juice, muriatic acid and pepsin should be administered after meals. If there be torpor merely, this may be overcome by the use of nux-vomica tincture, or the simple or aromatic bitters—these acting as local stimulants to the stomach-glands. If the appetite is languid and the stomach is equal to the digestion of the aliment taken, it will suffice to depend on the third group of remedies. A suitable supply of properly proportioned food is of the very highest importance. The albuminous or nitrogenous constituents—fresh animal food, eggs, milk, etc.—are the most necessary, but vegetables and fruits are also useful. If the digestive organs support food badly, it should be given in small quantity at short intervals, and, if solid food can not be managed by the stomach, beef-juice and milk can be given instead. The blood plasma may also be supplied directly by the rectal injection of defibrinated blood on the plan of Dr. Smith, of New York, which is a most important addition to our resources in the treatment of anæmia. A moderate quantity of alcoholic food is also highly serviceable—say, a tablespoonful of whisky three time a day—but it should always be remembered that a taste for alcoholic beverages is quickly formed under these circumstances. The medicines required are those actually used in reconstruction of the blood, viz., iron, manganese, and the phosphates. As iron and manganese exist together in the blood (1 to 40), and also throughout nature, it is very useful to follow this indication and administer them together. There is another view of the utility of iron—promulgated chiefly by Brown-Séquard—that it acts solely by increasing digestion, and that the food taken in increased quantity under its use contains sufficient iron to supply the requirements of the blood ; but the former view is that chiefly entertained. The saccharated carbonate of iron and manganese is an excellent preparation, or the dried sulphates of iron and manganese may be prescribed in pill-form, with or without extracts of nux vomica, gentian, or calumba. The question of the comparative utility of the vegetable or mineral-acid compounds of iron frequently arises. Notwithstanding the paradoxical character of the statement, it is generally true that the more irritating and astringent preparations are better borne, and they are certainly more effective. Next to iron and manganese are the phosphates, especially the phosphate of lime. In the anæmia of lactation there is a very marked deficiency in the quantity of phosphate of lime, and in all forms more or less reduction of the proper amount of this substance. The sirup of the lacto-phosphate is the best form for the administration of this agent, if well and genuinely prepared. Pyrophosphate of iron may be given with the phosphates, as compound sirup of the phosphates ; or the elixir of the phosphate of iron, quinine, and strychnine may be prescribed under the same indications.

When purpura, or the hæmorrhagic diathesis, or allied states of the blood exist, great advantage is derived from the conjoint administration of ergot or digitalis with quinine ; for iron is not well borne when the hæmorrhagic tendency exists, although the blood may be deficient in this constituent. Among the remedies for promoting the nutrition of the body, cod-liver oil takes a high place. It is usefully administered with the phosphates, especially in those cases in which anæmia is associated with impaired nutrition of the nervous system, and lowering of the general nutrition in cases of pulmonary disease. In the anæmia produced by phosphorus, carbonic-acid narcosis, coal-gas poisoning, etc., transfusion has been successfully employed. Uninjured new elements introduced into the veins, the condition of anæmia is at once removed. The operation of immediate transfusion of human blood is alone justifiable under these circumstances, for lamb's blood will not functionate properly. When the food is undergoing final conversion into blood, the oxygen of the air is necessary to complete the changes. Hence some exercise, short of fatigue, should be taken about three hours after the meals, for at this time the products of digestion are pouring into the blood, and then the oxygen is especially needed. Moderate exercise effects a proper distribution of the blood in the body, increases the absorption of oxygen, and the excretion of carbonic acid and urea. In proper limits exercise promotes the metamorphosis of tissue, and is therefore serviceable in anæmia, but, carried to fatigue, waste is greater than repair. The method of combined rest, massage, faradization, and forced feeding, practiced by Weir Mitchell,* is extremely useful in these cases, and will often succeed when other means fail.

CHLOROSIS.

Definition.—Chlorosis and anæmia are usually regarded as identical disorders, but they differ sufficiently to be treated separately. The peculiarities of chlorosis are simply referred to the sexual condition, and it is therefore, according to this view, an anæmia occurring in girls about the period of puberty. The term *chlorosis* relates to the peculiar tint the complexion assumes in this disease, and in common language it is designated "green-sickness."

Etiology.—Chlorosis is a disorder of the female sex almost exclusively, and those cases occurring in males are examples of modified anæmia. Puberty, or the period of sexual evolution, is the time of life when this disorder develops—from the fifteenth to the twentieth year. An inherited disposition seems to exist in many cases, for nothing is more common than the references of the mother to her own experience when the daughter betrays the first signs of the malady. The type of constitution which is thus transmitted is distinctly of lowered vitality—"the gelatinous descendants of albuminous parents" is the apt phrase descriptive of the constitutional state. These subjects are light, fair, full, round, but white, having blue eyes, soft tissues, and feeble muscles. Menstrual irregularities seem closely associated with chlorosis, either as cause or effect. According to Virchow, abnormal narrowness of the aorta is an important factor. If an hereditary predisposition exist, or congenital defects in the vascular system, the ordinary contingencies of social life may suffice to develop it—especially the cultivation of the emotional life—but it occurs quite independently of erotic sentimentality. On the other hand, this condition of the system comes on without any apparent cause, or spontaneously. Hammond, who has made an elaborate study of chlorosis ("Journal of Psychological Medicine"), maintains that it is an affection of the nervous system, the blood-changes being secondary.

Pathological Anatomy.—The body is fairly well nourished, and the subcutaneous fat pretty well distributed. The organs are generally pale. The serous cavities contain but little fluid, and there is no œdema of the inferior extremities. The most important change occurs in the blood, and consists in a diminution of the red corpuscles. This can now be readily determined by actual count, using the hæmacytometer, as modified by Gowers, for this purpose. As the iron of the blood is reduced in this disease, it is probable that the diminished staining power, which is so conspicuous an alteration, is due as well to diminution of the hæmatin as to loss of corpuscles. In chlorosis the albuminates and the leucocytes are not diminished, unless an anæmia develops in the course of the former, when the alterations peculiar to the latter are superadded. Neither is the volume of the blood apparently reduced. We owe to Virchow the important fact that in recurrent and persistent chlorosis, abnormalities exist in the vascular system: the aorta and arterial system, generally, are smaller in caliber, and thinner, the intima having a "trellis-like" arrangement; and the tunics of the vessels are affected by fatty degeneration in spots, and striæ of a yellowish color, especially the intima. These spots are found in greatest numbers about the origin of the ascending aorta, and on close examination are found to be a collection of minuter spots, each corresponding to a connective-tissue corpuscle, which is advanced in fatty degeneration. The heart may be normal, may be abnormally small, may be somewhat hypertrophied, but the alterations of this organ are not constant. The spleen, the lymphatics, and the marrow of bones, are not affected in any way.

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* "Fat and Blood, and how to make them."

Symptoms.—Girls about the period of puberty are the subjects of chlorosis. With or without disorders of menstruation, the affected person experiences a change in her feelings, and becomes morose and despondent, or capriciously vibrates from an extreme of high spirits to corresponding depression, but low spirits is the habitual state of the largest number. There is no reason to believe that erotic feelings are mixed up with the gloomy fancies which dominate the mind, but nymphomania is in rare instances présent as a symptom. Hysterical manifestations may also occur, but do not constitute a necessary part of the malady. As respects the actual condition of the sexual organs, there are two forms of derangement which happen in chlorosis: there are the amenorrhœic form and the menorrhagic form—cases in which the menstrual flow is absent; cases in which the flow is excessive. After an attack of menorrhagia, or after the failure of the flow to appear, the changes in the mental state above mentioned manifest themselves. Then the complexion changes. Fair-haired and white-skinned girls (blondes) become pallid, and waxy, and puffy, but without œdema; dark-haired and dark-skinned girls (brunettes) assume a muddy, grayish coloration, with bluish-black rings under the eyes; the sclerotic being pearly and glistening, and the mucous membrane of the mouth pallid. There is present, constantly, a strong feeling of fatigue, and the least exertion causes weariness, while strong muscular effort induces exhaustion. Muscular effort of any kind starts the heart into tumultuous action, and brings on difficult breathing and a sense of oppression. The anæmic *bruit* heard at the base, and over the great vessels, exists in chlorosis as in anæmia. The pulse is rather full, but soft, the action of the heart irregular, the breathing not rhythmical, and a dry, barking, or noisy cough is not unfrequently present. The appetite is usually capricious—now satisfied with difficulty, now indifferent to food, but characterized by sudden desire for unusual articles, or by craving for pickles, slate-pencils, chalk, etc. Attacks of cardialgia are frequent and severe, and may indicate the presence of a gastric ulcer—a not infrequent complication of chlorosis.

Course, Duration, and Termination.—The course of chlorosis is affected by the social circumstances, and the treatment still more, by the presence of the changes described in the vascular system. There are several important complications which affect the behavior of chlorosis. The first is anæmia, the development of which increases the gravity and adds to the duration. Phthisis develops in a considerable proportion of the cases, and in part doubtless because of the narrowing of the aorta. Perforating ulcer of the stomach is an occasional and very fatal complication. The explanation of its relation to chlorosis is, probably, the existence of fatty change in the intima of a stomach-vessel, thrombosis, and rapid solution of the mucous membrane. Chlorotic subjects—those affected with the changes in the tunics of the arteries, certainly—are very liable to attacks of endocarditis. Virchow, to whom we owe our knowledge on the subject, has further pointed out that during pregnancy, and in the parturient state, they are apt to suffer from ulcerative endocarditis of a most malignant character.

Paroxysms of hysteria and attacks of chorea are not infrequent, especially the former. Chlorosis is also a large and important element in the formation of exophthalmic goitre, but the cases are too rare to give this fact importance here. The duration of chlorosis is very uncertain. It is not a self-limited disease, and manifests no tendency to spontaneous cure. It may terminate in recovery, in partial recovery, or in some intercurrent malady, as pneumonia, typhoid fever, endocarditis, perforating ulcer of the stomach, cerebral hæmorrhage, etc. The prognosis is favorable for simple, uncomplicated cases, but must be guarded for cases which recur, as they may be examples of chlorosis with vascular changes.

Treatment.—As lessened hæmatin and hæmoglobulin is the essential element in chlorosis, the administration of iron is the main point in the therapy. The combinations of iron with a mineral acid (tincture of the chloride, sulphate, etc.) are usually more effective than the so-called mild preparations. The addition of manganese is useful, because of the intimate association of these minerals in the blood-globules. The utility of iron does not consist solely in supplying to the organism of the chloritic a material which is deficient, but in stimulating the appetite and the digestion, so that more food is taken and disposed of more easily. It follows that iron must be given in large doses in this disease, and experience is in harmony with theory on this point. Excellent results are obtained from the conjoined or simultaneous administration of iron and the phosphates—notably from the pyrophosphate of iron and lactophosphate of lime. Again, many cases do better—the majority, within my observation—by the combination of iron with some agent having the power to exalt the cerebro-spinal functions, as arsenic and strychnia. An excellent prescription, notwithstanding the chemical incompatibility, is the pil. ferri carb. with arsenious acid or arseniate of iron; or, Fowler's solution may be given separately, after the chalybeate. Strychnia, iron, and manganese sulphates can be given in pill-form. Hammond, influenced by his theory of the nervous origin of chlorosis, holds that arsenic is the true remedy, and his experience supports his theory. The author has seen the best results from a combination of iron and arsenic, and this fact he urges upon the attention of his readers. A generous diet, out-door air, and moderate exercise, are essential elements in the therapy of chlorosis. The combined treatment of rest, forced feeding, massage, and faradization, advocated by Weir Mitchell in these cases, seems to succeed in many wonderfully. The measures above recommended, combined with suitable hygiene, rarely fail, however, to effect a prompt cure. No treatment will accomplish more than a temporary cure in those cases associated with changes or abnormalities in the vascular system; for the chlorosis will recur from time to time, and possibly the case terminate at last with ulcerative endocarditis in the pregnant or parturient state.

PROGRESSIVE PERNICIOUS ANÆMIA—ESSENTIAL ANÆMIA—MALIGNANT ANÆMIA.

Definition.—By the term *progressive pernicious anæmia* is meant a form of anæmia of most severe character, progressive and fatal, and accompanied, toward the termination, by a fever.

Causes.—This disease occurs usually in women from fifteen to forty years, who have been repeatedly pregnant or subjected to debilitating influences, as uterine hæmorrhage, or to bad hygiene. It is not known why, in some cases, these etiologic factors will cause anæmia, and, in a few rare individuals, excite the far more formidable, indeed malignant, ailment.

Pathological Anatomy.—There is little or no emaciation due to the disease. There may be a good deal of fat under the skin, and the body may present an appearance of fullness and roundness, due to a general œdema; but usually the œdema is about the ankles. The skin may contain petechiæ of a purplish or brownish tint, scattered over the trunk and limbs. There may be ecchymoses, having the various colors characteristic of extravasated blood at different periods, and vibices, due to the same cause, and produced by pressure. There is more or less serum in the various cavities, and the organs generally are pale and bloodless. The changes in the heart and arterial system are the same as already described (see ANÆMIA), and consist in fatty degeneration of the cardiac muscles (papillary) and of the intima of the aorta and principal arteries. The alterations in the composition of the blood are also similar to those of anæmia, but they are more extensive and profound. The volume of the blood is lessened, the red corpuscles are fewer, the albuminates of the blood diminished, and the fibrin is deficient. There is no constant disturbance in the normal ratio of the white and red corpuscles, although cases have been reported in which the leucocytes were increased.

Symptoms.—The exact beginning of pernicious anæmia usually passes unnoticed; an unwonted paleness, a sense of fatigue on the least exertion, hurried breathing, and palpitation of the heart, at length attract attention. This may be entitled the chronic form. In a few cases, happening during pregnancy, the onset is rather sudden, and extreme pallor, palpitation, and breathlessness on making any effort appear within a short period. The progress is comparatively rapid in both forms after the symptoms are fully developed, and in a short time the weakness is such that the patient is confined to bed, is unable to rise, and faints on attempting to assume the erect posture. Various local hæmorrhages take place, as epistaxis, bleeding from the gums, menorrhagia, extravasations under the skin and into the retina. The hæmorrhages into the retina are very common, and consist, on ophthalmoscopic examination, of small, blackish, brownish, or yellowish-brown spots, or larger patches covering more or less of the fundus. They may, when very minute, not affect the vision, although present in great numbers; but an extravasation in the retina of considerable size obscures the field of vision correspondingly (Immermann). Small extravasations or larger hæmorrhages may take place in the brain, with the usual results. A constant symptom is fever, but it does not appear until near the end of the case, and does not pursue a definite plan or type. When death is imminent, the fever not only ceases, but the temperature declines below normal, falling to 95° Fahr., or even lower.

Course, Duration, and Termination.—Although pernicious anæmia has been separated from allied states, yet in its course and behavior it strongly resembles anæmia and chlorosis, especially the latter, or more closely a combination of the two. It seems, as it were, anæmia added to chlorosis, and the worst features of each fully developed. The duration is not self-limited, and hence varies greatly. The acute cases usually terminate within two months, but the more chronic ones continue for three or four months. The mode of dying is by exhaustion usually, but life may be unexpectedly terminated by sudden paralysis of the heart, or by cerebral hæmorrhage. In some instances the end has been reached by the condition known as "Kussmaul's Coma." Sudden unconsciousness occurs in consequence of cerebral capillary embolism (white-cell embolism ?), or from the development of acetone (acetonæmia).

Diagnosis.—Pernicious anæmia is distinguished from anæmia and chlorosis by the severity of the symptoms; from albuminuria by the absence of albumen from the urine; from leucocythemia by the normal condition of the spleen, liver, and lymphatics; from Addison's disease by the absence of the bronzing. The prognosis is highly unfavorable, no cases of cure having been reported.

Treatment.—There is no specific plan of treatment. The anæmic symptoms require iron; but, if hæmorrhages are occurring, iron must be discontinued, when arsenic, ergot, and quinia may be substituted. A generous diet and stimulants must be administered from the beginning. The best results have been obtained from the administration of phosphorus, and from arsenic given subcutaneously and in full doses.

THROMBOSIS AND EMBOLISM.

Definition.—By the term *thrombus* is meant the formation of a clot in a blood-vessel—an *ante-mortem* coagulation. The mechanism of its formation and the pathological changes associated with it are called *thrombosis.* A detached clot, or parts of a clot, or any new formation circulating in the blood-current, is designated an *embolus,* in the plural *emboli,* as fibrin embolus, fat embolus, pigment embolus, etc. The secondary obstruction and the changes consequent thereon, produced by an embolus, are known as *embolism*—as cerebral embolism, pulmonary embolism, etc.

Causes.—The process of coagulation of the blood consists in the precipitation and consolidation of certain of its constituents, which, under normal conditions, remain fluid. When a blood-clot forms, the fibrino-plastic substance acts on the fibrinogenous, the former contained in the blood corpuscles, the latter in the liquor sanguinis. This formation of fibrin, by the reaction between two other principles, is like the production of prussic acid by the reaction between amygdalin and emulsin, or of the volatile oil of mustard, by myrosin and myronic acid. The formation of fibrin, or the coagulation of the blood, only takes place in the vessels when there occurs a slowing of the current, or when there is a change in the parietes of the vessels. In diseases characterized by abnormal increase of the fibrin (*hyperinosis*), should the blood-current be much reduced in rapidity and force, coagulation will take place. Thus in *post-partum* hæmorrhage, a thrombus not infrequently forms in the pulmonary artery. When the *vis-a-tergo* is weak, and an obstacle is placed in the capillary region in front, thrombi may form in the veins next the capillary system—as, for example, in the pulmonary veins, in chronic interstitial pneumonia; in the renal veins, in parenchymatous nephritis, etc. Again, when vessels are divided, hæmorrhage is arrested by thrombi which close the divided extremity. Thrombosis, the result of changes in the tunics of the vessels, is more frequent in relation to disease of the arteries than of the veins. Formerly the notion was entertained that phlebitis played an important part in the process of thrombosis and embolism; that the intima was the seat of exudations and other products of inflammation to which the formation of a clot was immediately due, but it is now known that inflammation of veins is interstitial; that the tunica intima, deprived of its nutritive materials, undergoes necrosis, and becomes a foreign body, about which coagulation of blood takes place. This, however, is a comparatively rare cause of thrombus formation, as this process occurs in the veins. It is in the arterial system that those changes take place which enter so largely into the phenomena of thrombosis and embolism—the results of endocarditis and endarteritis. The formation of vegetations in endocarditis, especially on the valves, is a fruitful source of embolisms. In endarteritis slow degenerative changes occur in the walls of the vessels, the internal layer (intima) becomes involved—thickened, roughened, necrotic—and then thrombi form. Any foreign body, as a needle introduced into a vessel, will induce coagulation and the gradual formation of an obliterating thrombus. An embolus is formed when a portion of a thrombus, detached from the parent clot, enters the blood-current. The density of the clot and its position are important elements in the detachment of emboli. The softer the clot the more easily it is broken up, and, if situated near to the entrance of a communicating vein, the more certain a portion of it will be broken off from the main mass. The conical shape which the thrombus assumes, projecting beyond the point of attachment to the intima, and floating freely at its end, are physical conditions favoring its separation. Besides the action of these forces, emboli are detached by coughing, vomiting, sudden jars, straining muscular movements, etc. After fractures an immense number of fat emboli may enter the systemic circulation, and now and then a phlebolithe is a cause of obstruction; cancer products may penetrate the blood and be distributed widely; multiple embolisms may be caused by the entrance, from a depot of putrefactive matters, of putrid ferments; and pigment emboli may be a product of malarial fevers.

Pathological Anatomy.—Recent thrombi consist of soft, brownish-red coagula, either in the form of a plug which fills the vessel and entirely shuts off the circulation, or in a *plaque* or tablet attached to one side of the vessel-wall, permitting still a part of the blood to pass through. In the case of the latter, successive deposits of fibrin produce a stratified clot, which may ultimately obstruct the vessel. When a vessel is ligated, the clot formed does not extend beyond the first communicating vessel, but, when the thrombus is spontaneous, the coagulum may increase by successive deposition of material until it extends into a neighboring vessel. If a thrombus is suddenly formed, there will be a uniform distribution of the red and white globules throughout the coagulum; if slowly formed, the mass will have a stratified arrangement, due to the adhesion of the white corpuscles to each other, and their accumulation along the walls of the vessel, and on the surface of the clot, so that, when a section is made of a thrombus formed by successive deposition, it will be found to be made up by alternating layers of ordinary blood-clot and of white corpuscles. Thrombi are, therefore, of two kinds, stratified and unstratified. The first steps in the organization of a thrombus consist in a process of condensation: the liquid disappears, the red globules lose their color, and the mass contracts an intimate adhesion to the intima of the vessel. Vessels are formed by the union and canalization of migrated white corpuscles (Rindfleisch), and the remainder of the thrombus consists of a fine reticulation of fibers and corpuscles, but the corpuscles have usually disappeared at the expiration of two months. Softening of the clot begins in the oldest part. There is no attempt at organization, and the delicate reticulation of fibrin breaks up into a uniform granular mass. The red globules lose their coloring matter, and, mixed with the other contents of the thrombus, form a white or yellowish-white fluid having the consistence of cream, and an appearance like "laudable pus," but differing from pus in structure, for on microscopic examination it is seen to be composed of albuminous particles, fat-molecules, and altered blood-globules. While the interior of the thrombus presents this puriform appearance, the exterior may have the brownish-red of the clot, and there may be various shades of color representing various stages in the process of softening. When the process is complete there remains a puriform-like collection, in which no red globules remain undestroyed, and together with the white are transformed finally into fat-granules. An embolus derived from a thrombus will have the appearance belonging to the age and condition of the latter. The vessel in which it is lodged will be damaged at the point of lodgment, but in front and behind the embolus, will be healthy. The vessel may be completely or only partially obstructed. If completely, coagulation will ensue behind the point of obstruction forming a thrombus; if partially, successive depositions of coagulum will occur, and a thrombus will form about the embolus. The bifurcation of arteries is the usual point at which an embolus lodges. Its effects are not limited to the point of lodgment, but include the whole area nourished by the vessel, and the wider zone supplied by the branches remaining permeable. The part receiving blood through the obstructed vessel at once becomes anæmic; but the neighboring district is the seat of an active hyperæmia, which is designated *collateral hyperæmia.* One result of the increased pressure in this hyperæmic area is the rupture of small or large vessels and extravasation of blood. If the vessel obstructed is small and not a terminal artery, the anastomoses may be sufficient to supply the anæmic district. If, however, the compensatory circulation is insufficient or absent, the ischæmic part dies—undergoes *necrobiosis*, *gangrene*, or *necrosis.* The consequences following arrest of the circulation by an embolus depend largely on the position, still more on the size, of the obstructed vessel. Dry gangrene is produced by embolic blocking of a vessel of an extremity. In internal organs, especially the brain, centers of softening and fatty transformation of the tissue elements, and hæmorrhagic extravasations in the area of collateral hyperæmia, are results of embolism. Besides the hæmorrhagic extravasations, infarctions occur in the parenchyma of those organs supplied with Cohnheim's terminal arteries.*

Symptoms.—The position of a thrombus or an embolus exercises a most important influence on the symptoms caused by them. When a thrombus occupies a vein of an extremity, œdema of all the parts below is a result, and, if the obstructed vein is adjacent to important nerves, excessive pain, or troubles of motility, will also be present by reason of the pressure of the distended vessel. Gangrene is not a result, since the nutrition of the parts is accomplished, although feebly and imperfectly, but moist gangrene may be produced if other injuries are superadded—as erysipelas, traumatism, compression, etc. A cure in such a case is in part effected by the collateral circulation, but in a truer sense by the canalization of the thrombus. Notwithstanding the similarity in the symptoms, caused by thrombosis and embolism respectively, there is a great difference in the time at which the phenomena manifest themselves: the symptoms of autochthonous thrombosis come on gradually; of embolism suddenly, with shock (Wagner). Two classes of symptoms arise—affections of nutrition, from the simplest disorder up to gangrene, and functional disturbances, proper to the organ affected. These symptoms are not ascertained with the same facility in all situations. In the extremities, every step in the local process is easily followed and interpreted, but in internal embolisms only those symptoms due to perversion or suspension of function are recognizable. Embolic obstruction of a member is announced by a sudden and often intense pain and a chill, with numbness, loss or diminution of tactile sense, coldness, pallor of the skin, and a feeling of deadness and weight, and paralysis of the muscles; the pulsations wanting below, while above the obstruction they are full and strong. If embolic blocking of a vein in the brain, there occur defects of speech, hemiplegia, etc.; if of a pulmonary artery, sudden difficulty of breathing and sense of oppression, with, it may be, intense oppression and anxiety and death. Sudden attacks of amaurosis in puerperal fever, acute rheumatism, and pyæmia, are usually due to embolism of the central artery of the retina. Those organs not well supplied with nerves, as the liver, kidneys, and mucous membranes, do not offer distinct reactions on embolic blocking of their vessels, and hence the symptoms are obscure.* If the immediate danger of an embolic obstruction is past, even if the symptoms are very formidable, provided terminal arteries are not obstructed, they may disappear in some hours or days by establishing a collateral circulation.

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* Wagner, *op. cit.* "Untersuchungen über die embolischen Processe," von Dr. Julius Cohnheim, Hirschwald, pp. 112. Berlin, 1872.

Treatment.—As all the symptoms are due to the obstruction of vessels by a blood-clot, the point in the treatment of special importance is to effect a solution of this obstructing material. Theoretically, ammonia possesses a solvent power, and in its use the author has had most striking results in the case of thromboses and embolisms of the brain. To accomplish the purpose in view, ten grains of the carbonate of ammonia may be administered in a tablespoonful of solution of the acetate, three or four times each day. As, however, the action must be slow, the point of contact being small, the remedy must be very persistently employed. The iodide of ammonium may be administered in a solution with the carbonate also, and usually with good results. Other alkalies possess the same power, but to a less extent. The most generally useful is the phosphate of soda, in drachm-doses, three times a day, used for many weeks. As, however, prompt and speedy action is needed to avoid the serious structural alterations which occur so quickly, the ammonia preparations are preferable to any other having the same effects.

DISEASES OF THE HEART.

TOPOGRAPHY OF THE CARDIAC REGION.—METHODS OF PHYSICAL DIAGNOSIS.

The heart lies somewhat obliquely in the chest, behind the sternum, and extends downward to the left, so that the apex-beat is below and a little internal to the left nipple, and between the fifth and sixth ribs. The position of the heart varies somewhat with the position of the subject: it gravitates downward and forward when the posture is erect; backward and upward when the posture is recum-

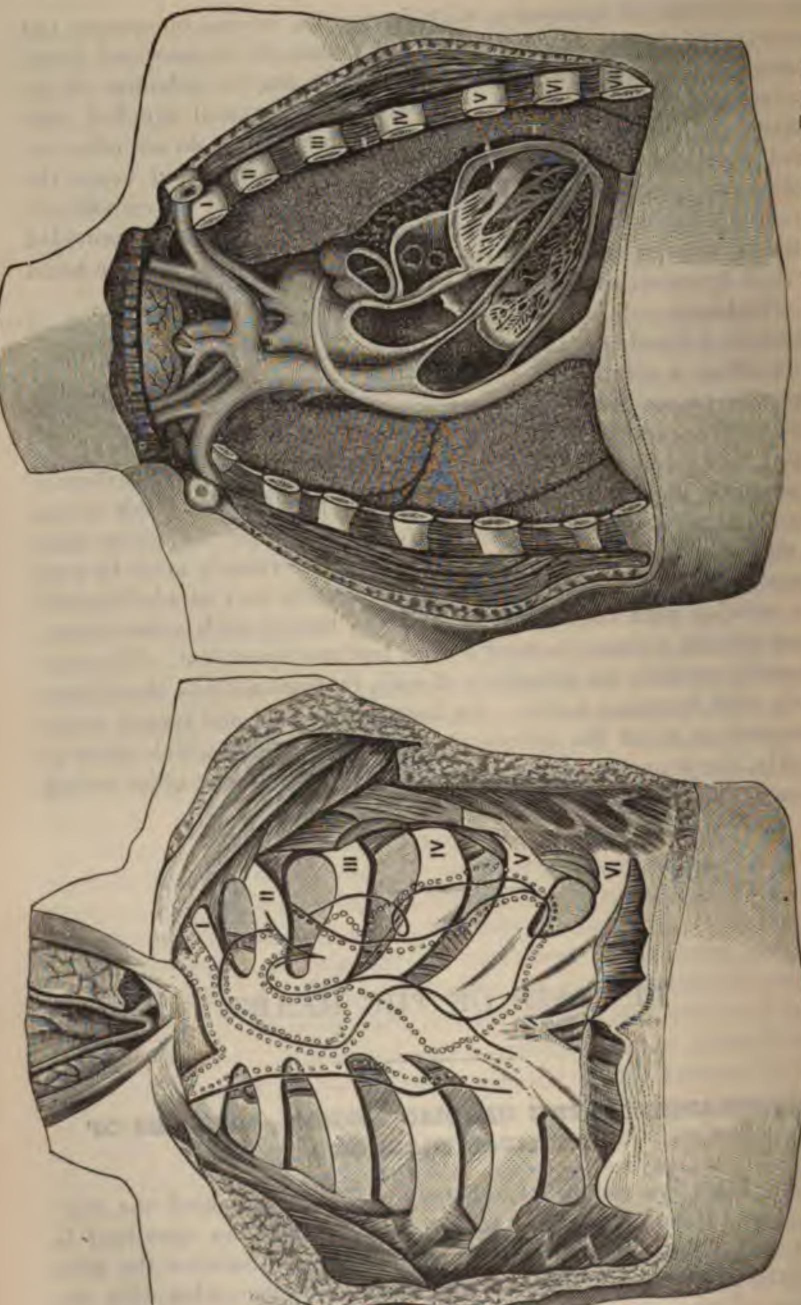

FIG. 19.—THE RELATION OF THE VALVES AND ORIFICES OF THE HEART TO THE RIBS, STERNUM, AND EXTERNAL SURFACE OF THE THORAX. Modified from Rüdinger.

bent. In general terms, the base of the heart is opposite the upper border of the cartilage of the third rib ; the most inferior part of the right auricle and the apex rests on a line parallel with the upper border of the sixth rib,* and in contact with the upper surface of the diaphragm (Figs. 19, 20).

It is important to note the position of the valves with reference to exterior points of the thorax.

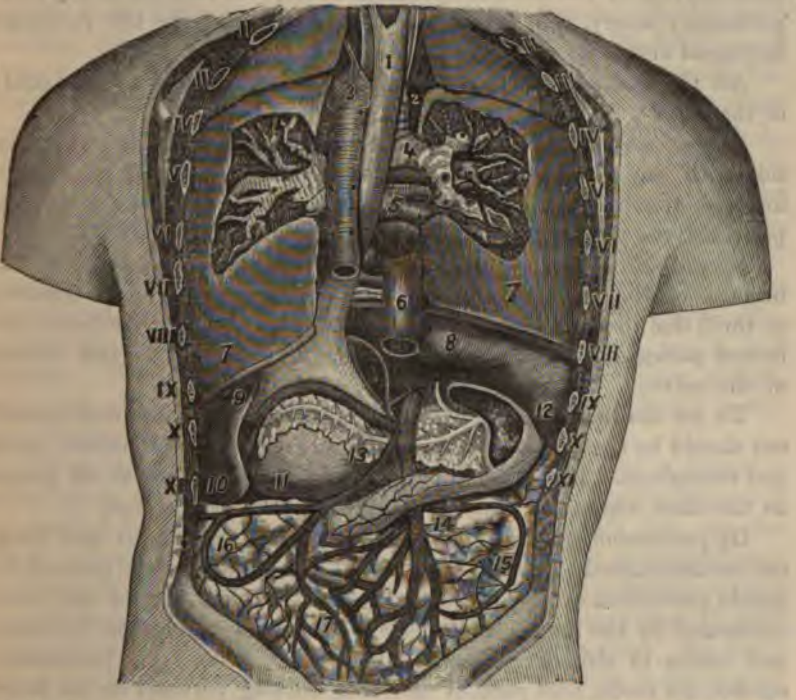

FIG. 20.—POSTERIOR VIEW.—1, Œsophagus; 3, Aorta; 4, Right Primary Bronchus; 5, Right Pulmonary Artery; 6, Ascending Vena Cava; 7, Lungs. After Rüdinger.

The aortic orifice lies under the junction of the cartilage of the third left rib with the sternum at its lower margin. The orifice of the pulmonary artery is at the left edge of the sternum, opposite the second intercostal space. The left auriculo-ventricular (mitral) orifice is somewhat less than an inch from the left margin of the sternum, and is beneath the third costal cartilage. The right auriculo-ventricular (tricuspid) orifice is under the center of the sternum, opposite the upper border of the fourth intercostal space. It follows that, if the stethoscope be placed on the junction of the third left costal cartilage with the sternum, its circumference will bisect each of the cardiac valves.

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* Henle's "Handbuch," Dritter Band, "Gefässlehre," p. 38 ; also Rüdinger, *op. cit.*

The ascending portion of the arch of the aorta extends about one fourth of an inch beyond the right margin of the sternum, and the vena cava lies half an inch to the right of the aorta, both vessels being behind the right second costal cartilage and the first and second intercostal spaces. The trunk of the pulmonary artery is beneath the sternum, to the left of the middle line, and extending somewhat more than half an inch beyond the left border of the sternum. The transverse portion of the arch of the aorta comes into relation to the right pulmonary artery, the left primary bronchus, and the left recurrent laryngeal nerve, all of which pass under it.

All the methods of physical diagnosis are applied to the study of the heart:

By *inspection*, the position of the apex-beat, protrusion or retraction with the apical impulse, area of impulse, abnormal position of impulse, abnormal pulsations in proper position, or pulsation in new positions, the venous pulse in the neck, etc., may be perceived.

By *palpation* can be ascertained the character and force of the impulse of the heart, the rhythm of its movements, friction fremitus or thrill due to the vibration imparted by the rubbing together of inflamed pericardial surfaces, and the purring tremor of certain lesions of the valves.

To get the best indications from palpation, the hand well spread out should be applied to the precordial surface, with the patient erect and recumbent. Palpation should, in turn, be practiced at all points on the chest where any abnormal pulsation may be detected.

By *percussion*, the area of cardiac dullness, superficial and deep, can be ascertained. By *superficial dullness* is meant that evolved by gentle percussion of the precordial region—of that part of the heart uncovered by the lung. This space is somewhat triangular in shape, and varies in size in different subjects. If only light percussion-strokes are made, that part of the lung coming forward on the heart is thrown into vibration, hence imparting the pulmonary quality to the tone. In determining the presence or absence of effusion into the pericardial sac, or hypertrophy, this superficial dullness has more significance than the deep. When strong percussion is practiced, the body of the heart is reached, the vibrations have a different character, and hence the production of *deep dullness*. The superficial dullness is not only more significant, but is also more easily determined. The condition of the pulmonary parenchyma, of the stomach and colon, and the degree in which the left lobe of the liver extends to the left, modify the deep dullness and increase the difficulty of defining it accurately.

The position, shape, amount, and degree of the deep cardiac dullness must be noted. The relation of these modifying circumstances to morbid states of the heart will be pointed out when the individual maladies are under consideration.

By *auscultation* we obtain the most exact information of the state of the heart. Certain facts regarding the normal condition of the organ must be clearly fixed in the mind in advance of the study of pathological states. That these may be the more readily comprehended and remembered, all statements not immediately utilizable are avoided.

The *cardiac cycle*, or the whole period of the heart's movement, has been variously divided, but it suffices for practical purposes to regard it as made up of four parts: 1st, the systole; 2d, the short interval; 3d, the diastole; 4th, the long interval. If the durations of these periods, respectively, are expressed in tenths, they have the following relations to each other: the systole, $\frac{4}{10}$; the first or short interval, $\frac{1}{10}$; the diastole, $\frac{2}{10}$; and the second or long interval, $\frac{3}{10}$. Certain sounds coincident with the systole and diastole are audible on application of the ear to the chest. These sounds are designated the *first* or *systolic sound*, due to the muscular contraction and the tension of the mitral and tricuspid valves, and the *second* or *diastolic sound*, due to the sudden stretching of the aortic and pulmonary valves. The first sound, audible with the greatest intensity in the mitral area, toward the apex, is rather dull, low pitched, prolonged, but is well defined. The second sound is much shorter in duration, clear, abrupt, and high pitched, and is audible with the maximum intensity in the aortic area—at the base.

The normal sounds of the heart are variously changed in disease—as to pitch, duration, quality, and rhythm. These modifications will be studied in connection with the maladies of which they are the signs. Doubling of the sounds, or *reduplication*, as it is entitled, may be a merely functional condition, or it may signify important structural changes. This phenomenon has been referred to a want of synchronism in the actions of the tricuspid and mitral valves. By Guttmann it is held to be the separation into distinct sounds of the several elements which, combined, make up the first or second sound, respectively—a lack of synchronism in the contraction of the papillary muscles acting on the segments of the mitral or tricuspid valve. This explanation is accepted by many as an adequate explanation of the phenomenon.

INFLAMMATION OF THE PERICARDIUM—PERICARDITIS.

Definition.—The term *pericarditis* means an inflammation of the pericardium. The inflammation may be limited to the parietal or visceral layer, or to a part of either, or it may involve the whole of both surfaces. In the former case, it is *partial* or *circumscribed;* in the latter, *general* or *diffused*. The inflammation may also be either *acute* or *chronic*.

Causes.—Idiopathic or primary pericarditis may arise from traumatism or from cold. In those cases supposed to be produced by changes of temperature there is usually, probably, a diathetic condition—as albuminuria—which escapes notice. Secondary pericarditis is more common, and is due to two causes: to an extension of inflammation from neighboring parts—pneumonia, left pleurisy, pulmonary tuberculosis, caries of the sternum or ribs, aneurism of the aorta, endocarditis, etc.; to the rheumatic dyscrasia. The dependence of pericarditis on rheumatism has been very differently stated by the different authorities. That in about one third of all the cases this complication arises is the opinion of Bamberger, and is doubtless a close approximation to the truth, but Thompson* says sixteen per cent. The severity of the cases, but not the position of the joints affected, has some influence in determining the frequency of the complication. The first attack is more liable to this complication; the second attack stands next. In Thompson's forty-three cases of pericarditis, twenty-five happened during the first attack and thirteen during the second. The author has seen three cases in which the pericarditis preceded the joint affection. Usually this complication arises during the period of greatest severity of the disease—during the second week, the favorite days being the ninth and tenth (Thompson). Pericarditis also occurs during the course of certain eruptive fevers, as scarlatina, variola, in puerperal fever, in albuminuria, scorbutus, etc., but there are no numerical data for an exact statement of the relative frequency. As regards the period of life in which pericarditis happens, there are differences in the two sexes—women being more liable during the period of puberty, thirteen to twenty, and men from twenty to thirty, the average being respectively nineteen and twenty-five (Thompson). Men are somewhat more liable to the disease than women, but the difference is slight.

Pathological Anatomy.—In the first stage of the inflammation there are two pathological conditions present: an alteration of the tissue, the seat of the inflammation; and an effusion into the pericardial sac. The inflamed membrane is marked by an arborescence of minute vessels, or is of a deep-red color, in consequence of the general stasis, and contains here and there spots of extravasation from rupture of over distended vessels. The membrane becomes dull, cloudy, and at first dry, and also swells from interstitial exudation, and its resistance is diminished by the separation of the connective-tissue elements. The stage of hyperæmia and suspended secretion is of short duration—lasting from a few hours to twenty-four, the shorter rather than the longer period. Rarely a case occurs in which there is no other than the interstitial exudation, no moist exudation on the surface, nor effusion into the cavity. Usually, after a variable period of a few hours, the membrane which was dry becomes coated, especially the visceral layer about the origin of the great vessels, with an exudation of fibrinous substance, having, it may be, a thin, pellicular character, or thicker and more consistent, but soon extending over both surfaces. Sometimes the exudation is reticulated, sometimes it forms conical or filiform projections—pineapple heart, *cor villosum*, *cor tomentosum*, etc. These peculiar appearances are due largely to the movements of the heart and the friction of the exudation on the two surfaces. When the exudation is sero-fibrinous, more or less straw-colored serum, having flocculi of lymph or masses of fibrinous substance floating in it, is contained in the cavity. Instead of being straw-colored the fluid may retain so much of the solid exudation churned up with it as to have a creamy consistence and a yellowish color; or it may have a reddish tint from a slight admixture of blood, or be composed largely of blood (hæmorrhagic pericarditis). The serous fluid may also have a yellowish tint from the presence of leucocytes, or the exudation may have from the beginning a purulent character. The latter is the case in pericarditis occurring during pyæmia, puerperal septicæmia, variola, etc. The hæmorrhagic exudation occurs in chronic alcoholismus and in scorbutus. There are, therefore, sero-fibrinous, hæmorrhagic, sero-purulent, and purulent exudations. A strictly serous exudation is found in general dropsy, in dropsy of the pericardium, etc., but not in true pericarditis.

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* "St. George's Hospital Reports," vol. iv, p. 31.

Effusions may be entirely removed, even those consisting largely of solid exudation. The fibrinous matter breaks up into a granular mass, which gradually becomes fatty; the cells also undergo a fatty metamorphosis; the watery part is quickly taken up and the fatty emulsion undergoes slow absorption. A complete restoration of the parts to the normal may ultimately take place, but this is an exceptional result. It is to be expected only when the exudation is largely serous, or when the fibrinous substance is deposited on a small extent of surface and is thin. Usually the watery part of the exudation is taken up; the migrated white-blood corpuscles in the mass of fibrinous exudation assume a fusiform shape, unite end by end, and form canals or blood-vessels, and thus an exudation becomes organized. The epithelium takes part in these changes, by the proliferation of its cells, and the mass of solid exudation is composed not only of fibrinous substance, but migrated leucocytes, and proliferating epithelium, mixed with a basis substance, composed of germinal matter.* Projecting masses of exudation, uniting from the two sides, form bands, which organize by the formation of vessels, and remain permanently. There may be a thin band or bands connecting the visceral and parietal layers, or larger and broader bands which, uniting, form subdivisions of the sac, or, the two surfaces may be glued together, entirely obliterating the cavity of the pericardium. The union may be so perfect that the most careful dissection can not separate them. Calcareous deposits may subsequently form in the exudation, or the whole of it may finally become so completely calcified, by the deposit of lime salts, that the heart is inclosed in an apparently bony case. The adherent pericardium is not unfrequently reported in medical journal literature as a congenital absence of this sac, and the calcification of an exudation, as the formation of a true bony envelope of the heart. The fluid exudation may persist notwithstanding the formation of neo-membrane and bands of adhesion, and it changes in quantity, now increasing while fresh deposits of fibrinous substance is occurring, now diminishing with a temporary amendment; sometimes assuming a hæmorrhagic character, but more frequently becoming purulent. The more solid and unorganized exudation, crossed here and there by bands of adhesion, assumes a grayish color, and undergoes ultimately a caseous transformation.

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* Rindfleisch, *op. cit.*, p. 265.

The muscular tissue of the heart becomes diseased by reason of the proximity of the inflammation—an acute myocarditis—which affects the muscular tissue in contact with the inflamed membrane. The muscular fibers become paler than normal, soften, and are infiltrated with fat-granules, so that the muscular contractility is impaired, and hence, if the lesion extends, the power of the heart will be greatly lessened. The extent of the pericarditis and the duration of the inflammation have a material influence on the extent of the myocarditis. In hæmorrhagic and purulent exudations, the damage to the heart is greater. The strain on the heart due to the increased exertion required in fever, and the compression of the exudation, interfering with the passage of the blood to the muscular tissue of the heart, also affect the nutrition of the organ, and favor degenerative changes. Endocarditis may result by an extension of disease from the inflamed pericardium, as has been experimentally and clinically established. In chronic pericarditis the myocarditis persists, the walls yield to the blood-pressure, and the cavities, the right especially, dilate.

Symptoms.—When an idiopathic pericarditis comes on, the initial symptoms occurring are those of any acute serous inflammation: *malaise*, chill, fever, increased respiration, loss of appetite, frequently nausea and vomiting. Pain of a dull, heavy character, or a feeling of soreness, is felt in the chest, but not invariably. Acute pain in the position of the pericardium is experienced only in those cases with pleuritis of the adjacent portion of the pleura, so that the real significance of any soreness or pain felt is ascertainable only on physical exploration. When pericarditis is secondary to an existing disease, there are no marked disturbances to indicate its onset—no distinctive increase in the temperature and pulse-rate, or in the respiratory movements, but there may be some præcordial anxiety and oppression, so that, in all cases of diseases in which inflammation of the pericardium is liable to occur, systematic physical exploration of the chest should always be practiced.

The fever movement in simple idiopathic pericarditis is of the remittent type, but in the secondary disease it does not modify that of the existing malady. The state of the circulation varies from a condition of high tension, with full, strong pulse, to great feebleness, low tension, and small, irregular, and unequal pulse. A weak, irregular pulse is characteristic only of cases with considerable effusion, with myocarditis, or exhausted by the severity and duration of this disease. The rational signs of pericarditis possess but little value; but the physical signs are highly significant. In the young, a small amount of effusion may render the præcordial space prominent, but, in adults, only a large accumulation will push out the intercostal spaces sufficiently to produce bulging, unless the lung is shrunken, or there are pleuritic adhesions so situated as to prevent the outward expansion of the pericardium. When there is any considerable distention of the sac and anterior bulging, the nipple of the left side is thrown up higher than its fellow of the opposite side. In consequence of the effusion, the sac of the pericardium is enlarged, and the mobility of the heart on changes of position is increased. Hence, on palpation, this increased mobility is ascertained by the different positions in which the apex-beat can be felt. When the effusion is sufficient to force the heart to a more horizontal position, the apical impulse is farther out and upward. As the effusion increases, filling the sac, the apical impulse becomes weaker and weaker, and is finally no longer felt, as the fluid is interposed between the apex-beat and the chest-wall. When the systole of the heart is weakened by myocarditis, or exhaustion, the apical impulse disappears earlier, especially if there be interposed a thick layer of soft exudation; on the other hand, the apex-beat will be felt longer when there is hypertrophy of the heart, and may not disappear at all if old adhesions keep the apex against the chest-wall. A change of position, as bending the body forward, may cause the apical impulse to be felt again when it had disappeared on the dorsal decubitus. On palpation, for a brief period may occasionally be felt a vibration of the chest-wall, due to the rubbing of the roughened surfaces together. To develop this sensation, firm pressure must be made in the intercostal space with the finger-tips. It is exceedingly rare for this friction fremitus to be strong enough to excite vibrations of the chest-wall, which may be perceived by the hand laid on the præcordial space. It is a rough, jarring, rasping sensation, similar to but quite distinct from the *frémissement cataire*, or purring tremor, and is not exactly isochronous with the cardiac systole and diastole, although a to-and-fro movement.

The area of cardiac dullness is increased when the effusion is sufficient in amount. The enlargement of the area of relative dullness is more important in a diagnostic point of view, because there may be no change in the absolute dullness, even when there is considerable effusion. The diminished sonority is first perceived at the sternal end of the third and fourth ribs—at the base of the heart. The dull space has a triangular form, with its apex uppermost and base downward—the right line of the triangle extending from the apex at the second rib and sternum, along the right border of the sternum, and even beyond, to the right sixth and seventh ribs and sternum; the base-line of the triangle passing through the seventh intercostal to the axillary border, and there intersecting the left line. When the effusion is extreme, the epigastrium is pushed outward by the descent of the diaphragm and the left lobe of the liver. The size of the triangular space is enlarged by sitting up and by bending forward. When the apex-beat can still be felt, and the area of dullness extends beyond it, this fact indicates that the sac of the pericardium is greatly distended, and consequently forced beyond the apex, and is therefore an important sign of effusion. A change in the position of the dullness may be slightly effected by changing the decubitus of the patient, the fluid obeying the laws of gravity. The pressure of the lung in the neighborhood of the pericardium is a necessary result of the accumulation of fluid; but this condensation is distinguished from effusion

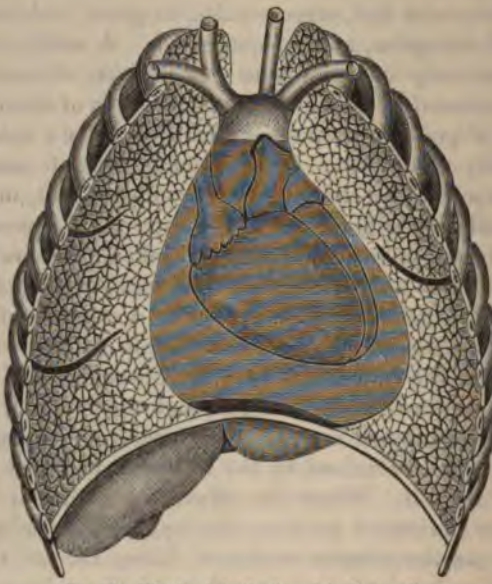

FIG. 21.—Effusion into the Sac of the Pericardium.

by the vocal fremitus, which is weakened or absent in the latter, but increased or normal in the former. In estimating the results of percussion, two sources of error may interfere: the dullness may be more extensive than the amount of the effusion warrants; it may be less. The first is due to adhesions which have the effect to retract the lung from the pericardium, and to push the heart forward, thus enlarging unduly the area of absolute dullness; in the other, the lung is attached anteriorly, and the heart lies deeply, and is still further depressed by the weight of the effusion. The pericardial *friction murmur* is the most significant of the physical signs of pericarditis, and is produced by the rubbing together of the two surfaces roughened by exudations, or by one roughened surface. This *bruit* makes the impression on the ear of scraping, grating, creaking, churning, and various modifications of these noises. They are, ordinarily, resolvable into three: the creaking of new leather, grating, or scraping. The sound may be partial or general; it corresponds to the seat of the exudation, and is not confined to the situation of the orifices of the heart, but is heard with the maximum intensity at the third intercostal space on both sides of the sternum. The area over which it is audible depends on the extent of the exudation. The *bruit* accompanies the heart-sounds, but is not confined to them, and extends into the interval, and may indeed occupy the whole revolution of the cardiac movement. Hence the term "*bruit de galop.*" Usually or frequently, the *bruit* is presystolic, systolic, and diastolic—the presystolic corresponding to the auricular systole, and the others to the systole and diastole of the ventricles. When there is no effusion (dry pericarditis), there will be usually no rational symptoms of the malady—nothing but fever, and the physical signs of pericardial inflammation.

The friction murmur, as well as the friction fremitus, occur early, and are recognized, if at all, within the first two days, and they persist for several days or weeks, according to the progress and amount of the effusion. They may decline in two or three days and disappear, as the effusion fills the sac and separates the two surfaces, so that friction is no longer possible. If the effusion is absorbed, then the *bruit* will become audible again. When the silence of the *bruit* is due to adhesions, there will be no return of it when it ceases. With the increase of the effusion the heart-sounds become weaker, and finally are no longer heard in some cases; but usually they continue to be audible, although very feebly. The character of the pulse, during pericarditis, has no special quality; it may be but slightly elevated above the normal; it may be very much accelerated; its rhythm may be much altered. At the onset of the inflammation, the pulse may be strong, the tension high; but this is not maintained, the pulse becoming weak, and the arterial tension low from depression of the vital powers and the occurrence of myocarditis. A large effusion exerts a mechanical pressure upon the great vessels within the pericardial sac —the aorta and pulmonary artery—and interferes with their proper filling. Also, as the veins can not empty their blood into the auricles fully, they are kept over-distended, and an abnormal fullness of the venous system in general is the result. Stasis of the venous system causes passive congestion of the lungs, bronchial catarrh, difficult breathing, cyanosis, and œdema. The venous congestion occurs in the brain, and is manifested objectively by headache, vertigo, epistaxis, etc.; in the liver, causing enlargement of the organ and hyperæmia of the portal system; and in the kidneys, inducing albuminuria. Irritation of the phrenic excites a most distressing hiccough. Difficulty of breathing, cyanosis, feebleness of the heart's action, are also produced by myocarditis, which is really an acute fatty degeneration. The heart's movements are not only feeble, but scarcely distinguishable; the pulse irregular, intermittent, feeble; the sounds of the heart are hardly recognizable, and the first sound is often absent; the temperature falls, the legs become œdematous, and death soon closes the scene. When severe dyspnœa and cyanosis come on in the course of pericarditis, they are more frequently due to the damage done to the heart's muscle than to the mechanical effects of the effusion. Again, the same symptoms, in a less extreme degree, however, may be due to nervous disturbance—to irritation of the pneumogastric and phrenic. Dysphagia may be caused by pressure of the effusion on the œsophagus, and aphonia by pressure on the recurrent laryngeal nerve.

Course, Duration, and Termination.—The course of pericarditis is not always upon a uniform plan, and there are peculiarities due to the causes and complications. Those cases arising in the course of puerperal septicæmia, scorbutus, or pyæmia, are shorter in duration, and greatly more fatal than those which are due to the rheumatic diathesis. The duration is influenced by many circumstances. In simple, uncomplicated cases, terminating in health, the effusion may be absorbed and recovery take place in from ten days to two weeks. When a case tends to recover, the severe symptoms subside, the fever and the difficulty of breathing cease, the appetite returns, and convalescence is established. When there is much effusion, and yet the tendency is toward health, the area of dullness lessens, the apical impulse returns, the friction murmur and fremitus reappear for a short period, the normal sounds are heard again, and, with these evidences of improvement afforded by the physical signs are also the rational symptoms of cessation of dyspnœa, of fever, and return of appetite. In other cases the improvement is partial; the rational and physical signs of pericarditis persist, and the subsequent history is that of chronic cardiac troubles. In other cases a fatal termination takes place early —in the scorbutic form with hæmorrhage in a few hours after the well-defined symptoms come on; in cases with large effusion, dyspnœa, delirium, etc., death will occur in a week or ten days; in cases with myocarditis and syncopal attacks, according to the age and other circumstances, a fatal termination may occur within the first two weeks. According to Thompson, the average duration of rheumatic pericarditis in St. George's Hospital is fifteen days.

Prognosis.—Simple cases of pericarditis, and rheumatic pericarditis, are not often fatal, and a favorable prognosis may be expressed in a very large proportion. As an intercurrent disease, coming on in the course of certain grave maladies, it is is extremely fatal. Among these may be mentioned scorbutus, pyæmia, puerperal diseases, Bright's disease, some of the eruptive fevers, pneumonia, etc.

Diagnosis.—The differentiation of pericarditis from endocarditis, hydropericardium, and left pleurisy, presents some points of difficulty. The separation of the endo- and exo-cardial murmurs is often an affair of extreme nicety. Dropsy of the pericardium is to be distinguished from the inflammatory affection by the absence of fever, local pain, and friction murmur. The character of the fluid in any case is to be determined only by the concomitant circumstances. If the patient is scorbutic, it is probably hæmorrhagic; if a subject of chronic alcoholismus, it may be hæmorrhagic; if the accompanying malady is pyæmia, or a septicæmic process, it is probably purulent; if rheumatism, it is sero-fibrinous; if albuminuria, serous. The differentiation of exo- from endo-cardial murmurs is based on the character, quality, seat, and persistence of the sounds. The friction murmur is a sound of rasping, of crackling; the endocardial murmur is softer, smoother. The friction murmur may be local or general, and has no constant relation to the orifices of the heart; the endocardial murmur is heard with maximum intensity within certain valve areas. The friction murmur is not regularly isochronous with the valve-sounds, or with the cardiac rhythm; the endocardial murmurs are usually systolic or disastolic, or coincide with the rhythmic movements of the heart. The friction murmur continues where it began; the endocardial murmurs are propagated in the direction of the blood-current—basal or apical. The friction murmur varies from one hour to another in intensity and extent; the endocardial murmurs remain constant. The friction murmur increases with pressure of the stethoscope on the chest-wall; the endocardial murmurs are not affected by pressure. The friction murmur increases in loudness with the upright position and bending forward; the endocardial murmurs are most distinct in the recumbent posture. The friction murmur disappears when the effusion reaches a certain amount, and reappears for a short time when absorption has taken place; the endocardial murmurs are permanent. The friction-sound of pleuritis is synchronous with the respiration; the pericardial is synchronous with the cardiac movements, or nearly so; suspension of respiration arrests the former, but does not affect the latter. When that portion of the pleura in contact with the pericardium is the seat of inflammation, a friction murmur, synchronous with the cardiac movements; in that case the distinction is impossible. In pleuritic effusion, as a rule, the dullness changes with the position of the patient, and in the upright position is over the inferior part of the thorax. In pleuritis with effusion, all voice and breath sounds disappear; in pericarditis, they are unaffected, except in so far as the lung is displaced by the enlarging pericardium. In hypertrophy of the heart, the action is heaving, and the apical impulse is strong; in pericarditis, with or without effusion, the impulse becomes weaker, and, as the effusion increases, the apical impulse will cease, or at least greatly diminish in force. In hypertrophy the absolute, in effusion the relative, dullness is increased; and, as has been pointed out, dullness exists beyond the apex of the heart when the effusion is large.

Treatment.—If the initial symptoms are recognized, a full dose of quinine sulphate (℈j) should be administered with a half grain of morphine, and the cinchonism should be maintained, by repeated smaller doses, for twenty-four hours or longer. When the evidence of effusion exists, there is no longer any indication for the use of quinine, since the inflammatory process has passed beyond control. The next object of treatment, and that which usually engages our attention at once, is the management of the exudation. There can be no question, at present, respecting the influence of ammonia salts in lessening the coagulability of the fibrinogenous substance. The carbonate should be given in solution of the acetate—five grains every two hours—when the exudation is forming, and to procure its disintegration and absorption, thus preventing adhesions. If the state of the patient will warrant, pilocarpine can be given with much advantage in most of the cases. Untoward results need not be feared if the proper dose of the remedy is not exceeded, and the mode of administration may be stomachal or hypodermatical—for there is little difference in the amount of the action in either way. As it exerts remarkable power in the removal of effusions and exudations, every case, resisting the more simple expedients should have the advantage gained by the use of pilocarpine. If the dose does not exceed $\frac{1}{5}$ grain of the hydrochlorate—and in susceptible subjects one-half ($\frac{1}{10}$) of this amount may suffice—no dangerous symptoms can arise from it in the case of adults. As, however, the production of physiological effects is essential to therapeutical action, the dose insufficient to cause symptoms can not do good.

When the initial symptoms make their appearance, if the patient is robust, six to ten leeches should be applied to the epigastric region; they should be allowed to fill and fall off, but the bleeding should not be encouraged. Dry cups may be applied to the same point, if the condition be that of debility. With or without previous abstraction of blood, if the patient is not depressed and the action of the heart feeble, an ice-bag should be applied to the præcordia during the initial period, but this expedient ceases to be useful when there is much exudation, and may be very injurious if the heart is weakened by myocarditis. When the time comes for the removal of ice, good results may be expected from the application of flying-blisters. As a condition of quietude of the diseased organ is a measure of the highest utility, remedies which slow the heart are necessary. Aconite-root tincture and veratrum-viride tincture may be given to quiet the heart before considerable damage has been done. When, however, the heart begins to flag, remedies of a depressing kind are not suitable, and then digitalis becomes extremely serviceable, not only to lessen the work of the heart, but to promote absorption. The infusion is the best form, and it should be given in a tablespoonful-dose every four hours. The absorption of a pericardial effusion may be hastened by the use of pilocarpine—so administered as to act freely on the skin. But pilocarpine is too depressing a remedy when the action of the heart is feeble and the pulse is small and irregular. Stimulant doses of quinine and alcoholic stimulants are very important when the powers are failing and syncopal attacks are occurring. Mechanical means are proper when the effusion into the pericardial sac is great and does not yield to the remedies proposed. Paracentesis of the pericardium has now been performed many times with success, so that it can no longer be regarded as a doubtful experiment. The hypodermic syringe may be used to ascertain the character of the effusion. The needle, as in the operation for capillary puncture, is inserted close to the border of the sternum, in the fifth intercostal space. The operation of paracentesis is required when the effusion is great, or when it is purulent. If the effusion returns repeatedly, it is safe practice to inject the tincture of iodine (ʒ ij—℥ iv) to prevent the reaccumulation. If the contents of the sac are purulent, the iodine should be used more freely (ʒ ij of the tincture, ℥ ss potassium iodide, and ℥ iv water). To avoid wounding the heart, the patient should be recumbent when the puncture is made. The disadvantages of the operation are, that it is rarely curative; that it has caused a pneumo-pericardium; that the fluid is quickly replaced, because of the lessened extravascular pressure; that hæmorrhages take place by rupture of the thin-walled vessels of the neo-membrane. Better results are claimed from the operation of paracentesis when a part of the fluid is drawn at a time, rather than all at once.

ADHESIONS OF THE PERICARDIUM.

Adhesions of the two pericardial surfaces are results of pericarditis. They occur in a variety of forms: as narrow bands, as membraniform partitions, dividing the cavity into several smaller cavities, and sometimes these secondary sacs contain exudation, in the form of a caseous mass, or dark-brown deposits, a product of altered blood. The adhesion may be total, so that after some years no line of union can be made out between the two surfaces. The mass of exudation uniting the surfaces may be converted into an apparently bony case enveloping the heart by calcareous deposition. Bands of adhesion may exist externally to the pericardium, and unite this membrane to the neighboring pulmonary pleura, to the pleura costalis in front, to the mediastinum, etc. As has been pointed out in the preceding chapter, an inflammation of the pericardium leads to acute myocarditis—an acute fatty degeneration of the muscular tissue. Hypertrophy and dilatation are among the results of adhesions. Opinions are divided as to the precise part played by the adhesions, but there can be no doubt that atrophy with hyperplasia of the connective tissue are results of the myocarditis, which, in turn, induces dilatation of the cavities. When the cavity of the pericardium is obliterated, and adhesions have been contracted to neighboring parts also, the heart works to great disadvantage; but the most serious result is the interference with the nutrition of the organ. On the other hand, there may be entire adhesion of the two pericardial surfaces, and the heart be not at all incommoded.

Symptoms.—The disturbances produced by adhesions are manifested in rational and physical signs. The propelling power of the heart being diminished, stasis takes place in the right cavities, in the lungs, and venous system generally. There are therefore constantly present bronchial catarrh; difficulty of breathing; swollen liver and spleen; gastro-intestinal catarrh; urine scanty, high-colored, and albuminous; veins full, face cyanosed; general dropsy. The apical impulse is either wanting entirely, or is a mere tremor; the pulse is rather quick, but low in tension, and the volume varies in different beats. These rational symptoms are chiefly indicative of the degeneration and atrophy which have occurred in the heart-muscle. Other symptoms are caused by adhesions. One of the most important physical signs of pericardial adhesions is a depression with the systole of the heart at the place of the apex-beat. Instead of an elevation of the intercostal space when the apex of the heart is tilted against it at the time of the systole, there occurs a *depression, or drawing in of the chest-wall.* There may also be, at the left of the sternum, several small depressions or "pittings" in the intercostal spaces. These depressions are frequently due to pericardial adhesions of the two surfaces, and to the parietal pleura; but they may occur independently of this, as has been demonstrated by Friedreich, produced by causes which obstruct the downward movement of the heart toward the left, and the tilting of the apex upward, the lungs at the same time not coming forward sufficiently. A diastolic elevation of the chest-wall is the compensatory sign of the preceding elevation. When the force producing the other ceases to act, there is a rebound of the chest-wall, which, if not visible to the eye, may be felt on palpation. These two signs are highly significant, but their absence does not negative the existence of pericardial adhesions. It has already been stated that the area of absolute dullness is increased in those cases of adhesions which fix the heart against the chest-wall, and do not permit the organ to fall back, while at the same time the lung is prevented coming forward. If the heart is so fixed in position by adhesions, and is at the same time hypertrophied, and if the pericardium be adherent to the chest-wall, and to the spine behind, there must, of necessity, be produced the *systolic depression*. When the *diastolic rebound* ("diastolic concussion") occurs, a synchronous or diastolic collapse takes place in the cervical veins. Much distended during the systole, they suddenly subside and even disappear during the diastolic rebound, for during this act the chest is expanded and the blood is drawn into the cavity. The importance of pericardial adhesions depends much less on the adhesions than on the changes in the heart-muscle. Adhesion bands connecting the two surfaces may exist without injurious effects. When hypertrophy takes place compensation ensues, and the heart is equal to its duties for many years. On the other hand, when the heart-muscle undergoes atrophic degeneration, its propelling power is insufficient, venous stasis and dropsy follow, and then a fatal termination is near. The *treatment* in these cases must be directed to the nutrition of the heart-muscle. Rest must be enjoined; the appetite and digestion must be improved by bitters, mineral acids, and the ferruginous tonics. The heart must be toned up by digitalis and iron, and by the judicious administration of quinia and morphia—the latter in minute quantity ($\frac{1}{16}$ of a grain). The author has seen the greatest advantage from the use of sulphate of iron (gr. j), sulphate of quinia (gr. ij), sulphate of morphia (gr. $\frac{1}{20}$), and digitalis (gr. j) in pill-form, three times a day.

HYDROPERICARDIUM—DROPSY OF THE PERICARDIUM.

Pathogeny.—By *hydropericardium* is meant an accumulation of water in the sac of the pericardium without the occurrence of inflammation. After death, especially from chronic wasting diseases, there will be often found in the sac an ounce or two of fluid, poured out at the time of the death-agony and immediately after. In dropsy, properly speaking, the quantity of fluid may reach to one or two pints. It is a clear, yellowish, or straw-colored serum, usually, but it may present a somewhat turbid appearance from the presence of cast-off epithelium, or a bloody appearance derived from hæmatin. This fluid has the composition of the blood-serum, and its alkaline reaction, but does not contain the same relative proportions of its constituents. The albumen is less than in the blood-serum, and also some of the salts; but it contains the fibrinogenous substance which sometimes coagulates when exposed to air. Urea is found in this fluid in renal diseases, and it is stained with bile-pigment in cases of jaundice. The fluid, if large in amount, dilates the sac, and its walls become thinned by the pressure, and often present a sodden appearance when there has been a protracted contact of the fluid with the endothelium. The subserous fat is absorbed by the pressure, and the areolar tissue is infiltrated with fluid.

The causes of hydropericardium are twofold : mechanical and dyscrasic. Diseases or neoplasms,* that interfere with the return of blood through the veins, as tumors, obstructive pulmonary disease, emphysema, and dyscrasia, such as Bright's disease, cancer, and tuberculosis, are the principal etiological factors.

Symptoms.—A small quantity of fluid will not produce sufficient disturbance to cause recognizable symptoms ; a large effusion will be recognized by the rational and physical signs, such as were described under pericarditis, with effusion. There is, of course, no friction murmur. The apical impulse becomes more and more feeble as the effusion increases, and it ultimately ceases to be felt. The heart-sounds grow more and more feeble, and may disappear entirely. The area of relative dullness greatly increases and extends finally beyond the region of apex-beat, and has the characteristic triangular form of dullness from effusion. The diagnosis of hydropericardium, from the effusion of pericarditis, rests entirely on the history—the latter being due to inflammation, the former not. The prognosis of this malady is serious, not wholly because of the fluid, but on account of the conditions associated with it. The treatment is directed to the removal of the fluid, and consists in the use of eliminants and mechanical means ; purgatives, diaphoretics, and diuretics are employed to procure absorption. Saline purgatives, compound jalap powder, elaterium, are given to diminish blood-pressure and the quantity of fluid ; squill, digitalis, and cream-of-tartar, to excite diuresis ; warm baths and pilocarpine to stimulate the skin. These means may be entirely successful in some few cases in Bright's disease, for example, but will have but little effect in cases of emphysema, tuberculosis of the lungs, and when the effusion is due to the pressure of a tumor. Aspiration is proper when life is threatened by the extent of the effusion, but there is danger of exciting pericarditis and of the admission of air.†

HYDROPNEUMOPERICARDIUM.—This form of disease differs from the preceding in that air or gas, as well as fluid, is present in the cavity. The fluid, when gas is also present, is composed of some decomposing exudation, of pus, or of blood. The first named is derived from pericarditis, the result of traumatism, or excited by an ulceration penetrating the cavity from the neighboring parts. The symptoms are physical. The space of absolute dullness is occupied by a tympanitic sound, except at the base, where it is dull from the presence of fluid. Change of the patient's posture alters the position of the dullness. The heart-sounds and the apical impulse are sometimes feeble and may not be perceptible, but are usually loud, splashing, and prominent. A peculiar, clanging, metallic character is imparted to the heart-sounds. The friction murmur has a rough, rasping, metallic resonance. Very remarkable sounds are produced by the churning of the liquid and air together by the heart-movements, and are designated "the water-wheel sounds." The functional disturbances produced by hydropneumopericardium are those of pericarditis, and need not therefore be recapitulated. The prognosis is grave ; yet, of fourteen cases collected by Friedreich, only ten proved fatal. It has usually been regarded as more fatal than these figures indicate. It is probable that some of them were examples of the admission of air merely, and were not produced by the gas of decomposition. The treatment is that of pericarditis. The presence of decomposing materials, or such an excess of gas or fluid as to exercise dangerous compression, justifies the employment of the aspirator, and washing out the sac with an iodine solution.

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* "Transactions of the Pathological Society of London," vol. xxii, p. 123.

† Roberts, "Paracentesis of the Pericardium." Philadelphia, 1880. An excellent work.

INFLAMMATION OF THE MUSCULAR TISSUE OF THE HEART —MYOCARDITIS.

Definition.—The cardiac muscle is subject to attacks of inflammation, as muscular tissue in other situations. The term *myocarditis* includes several morbid conditions of an analogous kind, but different in seat and also in progress.

Causes.—The male sex is more liable than the female. The acute form is more common before than after thirty years of age. Myocarditis may occur during intra-uterine life, and then preferably on the right side, setting up important changes. It is supposed that chilling the body, suddenly, when in a warm and perspiring state, will cause this disease ; again, violent muscular exertion is said to have excited inflammation ; but these are very doubtful causes. In fact, nothing is definitely known of the influences setting up such a morbid process in the heart-muscle. As regards the secondary diseases, our information is more definite. It has already been pointed out that myocarditis is a result of pericarditis, the inflammation extending by contiguity of tissue. It results from valvular lesions also, and may be secondary to the acute infectious diseases—as typhoid, pyæmia, scarlet fever, etc. Inflammation and abscess may be the result of embolic obstruction of the coronary artery.

Pathological Anatomy.—The muscular tissue itself, or its intervening connective tissue, may be the seat of the inflammatory action; consequently there are two forms—parenchymatous and interstitial.

The *parenchymatous* may occur in two forms; in isolated patches, or generally diffused. When a large part of the organ is attacked, there is a marked change in its appearance. The muscular tissue has a reddish color, is puffy in appearance, and the pericardium is spotted with points of ecchymoses, is cloudy, and coated here and there with a delicate exudation. The muscular tissue, on microscopical examination, is found to be cloudy, granular, and swollen, and the striæ indistinct or absent, or the fibers are broken up into granular fragments, are crowded with fat-granules, and ultimately are replaced by rows of fat-granules. When the change is far advanced, the muscle is brownish in color, and almost or quite pulpy in consistence. This change may extend over large parts of the organ, or may be confined to spots or isolated patches, and certain parts of the heart are especially apt to suffer, as the apex of the left ventricle, and, at the base, the posterior wall; next, the aortic valves adjacent to the septum, then the papillary muscle, and, on the right side, the muscular trabeculæ.

Interstitial myocarditis also occurs in two forms: the suppurative and the sclerotic; the former being acute, the latter chronic. Suppurative interstitial myocarditis usually coincides with the parenchymatous; and, between the muscular elements disintegrating with acute fatty degeneration, is seen more or less extensive dissemination of pus, or distinct and isolated collections, or abscesses. When the suppuration is due to emboli, the purulent collections are small, and there are usually several; when the result of interstitial inflammation, there is usually a single large one. An abscess may rupture outwardly into the sac of the pericardium, or inwardly into the cavity of the heart. If situated in the septum, by the discharge a communication is established between the two ventricles, or it may cause a rupture of a segment of the semilunar valve, an example of which has fallen under the author's observation. Again, an abscess in the walls discharging into the ventricle, forms a sac which, bulging outwardly under the blood-pressure, becomes an "aneurism of the heart," so called.* The interior of such a sac becomes lined with successive layers of fibrin, which protects the cavity from rupture, but only for a brief period. When an abscess discharges into the pericardium, a fatal pericarditis results; when the purulent matters and shreds of broken-down tissue enter the ventricular cavity, they produce the disastrous results of multiple embolisms.† Rarely, the pus is absorbed, and a mass of con-

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[*] "Transactions of the Pathological Society of London," vol. xix, p. 149 (with plate).

[†] Ibid., vol. xx. "A case of abscess of the heart bursting into the left ventricle." Boy of eleven years had a fall injuring his shoulder; delirium, wakefulness, fever, and a very rapid pulse, but no cardiac symptoms succeeded. He died on thirteenth day.

nective tissue and a puckered cicatrix remain to indicate the nature of the disease.

The *chronic interstitial myocarditis* is sometimes called sclerosis of the heart, or fibroid degeneration (Legg) of the heart. It consists in a proliferation—an overgrowth—of the connective tissue and an atrophy of the proper muscular elements. There may be small bands of connective tissue stretching between the muscular fibers, or larger, firm bands, or indurated masses, which take the place of muscular tissue entirely. These bundles or masses of connective tissue occur in the papillary muscle of the left ventricle and in the walls, but more toward the apex than at the base. Two evils result from the presence of these bands and masses of connective tissue and from the resulting muscular atrophy: the propelling power of the heart is reduced and stasis occurs in the venous system; the walls yield at those places composed of the connective tissue, and form the so-called "partial aneurism of the heart." It is especially at the apex of the left ventricle (eighty-five in eighty-seven cases) that these aneurisms form. They vary in size from a pigeon's to a hen's egg, are irregular and divided by partitions and often have diverticula attached, and they contain old deposits of fibrin and recent soft coagula. The walls of these partial aneurisms are composed of the sclerotic material, the endocardium, and the visceral layer of the pericardium with, it may be, the parietal layer attached.*

Symptoms.—The existence of myocarditis can hardly ever be anything but a presumption, based on negative rather than positive signs. If maladies are present, as rheumatism, pyæmia, puerperal fever, etc., in the course of which myocarditis may be expected, if the symptoms of cardiac failure come on suddenly, and if they can not be referred to an endocarditis or pericarditis, then the existence of inflammation of the heart-substance may be suspected. When this disease occurs as secondary to rheumatic endo- or pericarditis, the patient passes rapidly into that condition of profound adynamia known as the *typhoid state.* When an abscess discharges its contents into the cavity of the heart, the symptoms of multiple embolisms are produced; there are repeated violent chills, very high febrile temperature, profuse sweats, icterus, swollen spleen, albuminuria, delirium, or the disturbances due to embolism of the cerebral vessels, etc. The yielding of the sclerosed tissue and the formation of the so-called aneurisms are announced by failure of the heart; the pulse becomes thready, the lips blue, the face anxious, livid, and cyanosed, the respiration embarrassed, the surface cold, the weakness extreme, death occurring in a short time in syncope.

Those cases of myocarditis in which the symptoms of embolism are wanting, and aneurismal dilatations have not occurred, are characterized by the presence of the following signs: The movement of the heart is feeble, and the apical impulse unfelt; the pulse is small, weak, irregular, and intermittent. The great diminution which has taken place in the propulsive power of the heart manifests itself in stasis, pulmonary engorgement and œdema, cyanosis of the face, swollen veins, vertigo, delirium, etc. In the so-called chronic partial aneurism, there may be no symptoms for a time to indicate the existence of the lesions. We have here the same groups of symptoms, due to the diminished propelling power of the heart, as in the preceding paragraph, when sufficient damage has been done to cause yielding of the cardiac wall.

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* Ponfick, Virchow's "Archiv," Band lviii, p. 528.

Course, Duration, and Termination.—The course of the acute form of myocarditis is very rapid, and the duration from two or three to eight days, but some of them terminate in a few hours. Death may be due to rupture of the heart, to cerebral emboli, to pulmonary œdema, to paralysis of the heart, etc. Chronic myocarditis pursues a very latent course. The development of the lesions may be slow, and hence the duration may be prolonged, but not indefinitely. Dilatation of the cavities, feebleness of action, and stasis, will bring on fatal lesions in a few months, or, at most, a year or two.

Treatment.—The treatment must be largely symptomatic, and for parenchymatous carditis is to the last degree inefficient, since the causes are not to be removed. Interstitial inflammation, like the same disease elsewhere, is little influenced by remedies. Minute doses of chloride of gold, or of corrosive chloride of mercury, quinine, and digitalis, offer the best prospect of improvement. The utmost quietude of mind and body must be maintained. A generous diet and means to promote digestion are necessary to improve the quality of the blood.

FATTY DEGENERATION OF THE HEART.

Definition.—A distinction must be drawn between fatty degeneration and fatty substitution: the former implying a change in the structure of the muscular tissue; the latter, a displacement of the muscular tissue, in which atrophy of the muscular elements may take place, by the pressure.

Causes.—The nutrition of the heart is impaired by a variety of causes, intrinsic and extrinsic. Among the intrinsic are pericarditis and myocarditis, which set up an inflammation of the heart-muscle; diminished blood-supply due to atheroma; compression, etc., of the coronary arteries; fat substitution, which, encroaching on the proper tissue of the organ, causes absorption, etc. Among the extrinsic causes are impaired nutrition in general, originating in various ways—cancer, tuberculosis, scrofula, prolonged suppuration, prolonged lactation, etc. Most of the foregoing causes induce atrophy by setting up a fatty degeneration. Anæmia, especially when extreme and long-continued, has a strong tendency to induce this change. This has been demonstrated experimentally by Perl,[*] and clinically by Ponfick[†] and others. In the various causes above given it is the condition of anæmia induced by them which is responsible for the changes in the heart's muscular tissue. Infectious diseases, fevers, and certain poisons, notably phosphorus and alcohol, bring on fatty degeneration. The same result is produced by the mineral poisons in general, but to a less degree, and some other substances. Fatty deposition sometimes takes place to a dangerous extent in the obese, along the sulcus, and penetrating to the endocardium. Furthermore, in the anæmia of the obese, sometimes a very marked condition—fatty degeneration of the heart-muscle—comes on.

Pathological Anatomy.—The change may be general or diffused, or *exist in spots and patches.* The color becomes yellowish, the tissue soft and easily torn, and on the touch makes, in advanced cases, a distinctly greasy impression. The initial change is in the primitive bundles, which become cloudy, granular, and their striæ disappear. Minute oil-globules appear, and are soon seen in rows, but they presently coalesce ; large globules are formed, and nothing is then visible in the sarcolemma but a multitude of fat-drops. With this change in the fibrillæ of muscle, an œdematous condition of the sub-serous connective tissue occurs, and the nutrient vessels are advanced in calcareous degeneration. The fatty change may occupy the walls of the left ventricle, or be confined to isolated patches here and there in the walls of the heart, the papillary muscle, the trabeculæ, the septum, etc. In the cases of fatty substitution, the whole heart may be enveloped in a dense layer of fat, which also pushes its way into the muscle, following the inter-muscular planes and the connective tissue, causing such compression that the muscular fibers undergo atrophy, and are pale, thin, and wanting in contractile power.

Symptoms.—Weakening of the heart, produced by fatty change in its muscles, causes the disturbances due to ænemia of the organs and to venous stasis. The rational are more significant than the physical signs. On palpation, the apical impulse is weak. On percussion, there is nothing distinctive, except an increase of the area of absolute dullness, if the organ is enlarged by dilatation of its cavities. As there is venous stasis, and as the right cavities yield more than the left, the area of dullness is increased over the lower end of the sternum to the xiphoid appendix. On auscultation, if there be fatty degeneration of the papillary muscle, a systolic murmur is audible in the mitral area. The sounds of the heart are dull, confused, almost inaudible, and there is often a failure of synchronism in the closure of the valves, causing double sounds. The pulse is small, irregular, intermittent, weak, and easily compressed, and may be very slow, falling to forty, often even as low as twenty; but this is exceptional. A very formidable symptom, which, however, occurs under other circumstances, is a peculiar alteration of the respiratory rhythm, known as the Cheyne-Stokes breathing, in which at intervals the respiration becomes slower and shallower, until finally it seems to cease—is suspended for some seconds, half a minute, for a minute—and then is resumed, slow and shallow, but gradually attaining its normal amplitude. This may be kept up for some time, then disappear, to occur again. The diminished propulsive power of the heart, causing anæmia of organs, induces characteristic symptoms. Sudden anæmia of the brain, faintness, and actual fainting, often occur on rising up suddenly from a recumbent posture, stooping, turning around quickly, etc. These subjects experience constantly, or nearly so, a sense of fullness and distention about the ensiform cartilage or lower sternum, which is associated with præcordial anxiety, and they have attacks of angina pectoris.* They experience difficulty of breathing on slight exertion, and can not ascend elevations or stairways without experiencing great distress. The veins of the neck are habitually distended, and the countenance looks dusky and anxious. The legs become œdematous; next, the body generally; the liver enlarges, ascites forms, the urine becomes albuminous, etc.

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[*] "Ueber den Einfluss der Anämie auf die Ernährung des Herzmuskels," Virchow's "Archiv," Band lix. p. 39.

[†] Ponfick, "Berliner klin. Woch.," "Ueber Fettherz," Nos. 1 and 2, 1873.

Course, Duration, and Termination.—Acute fatty heart, produced by the action of poisons, terminates early; but the cases due to the ordinary causes proceed more slowly, and may last during several years. Their development is obscure, and there are no pronounced symptoms until those of failing heart come on. The termination is in general dropsy, or death is caused by œdema of the lungs, or takes place suddenly by paralysis of the heart, or by rupture of the organ.

Diagnosis.—If the causes of fatty degeneration have existed, and symptoms of cardiac weakness come on slowly, the existence of fatty heart may be regarded as probable, but the diagnosis is largely the balance of probabilities, and is not to be arrived at by exclusion with certainty.

Treatment.—As anæmia plays so important a part in the causation of fatty degeneration of the heart, the treatment should be directed to the enrichment of the blood. Iron, manganese, and strychnine (the sulphates), is an excellent combination. The author has seen good results from the phosphate of iron, quinine, and strychnine, in the form of the elixir. Jaccoud prefers caffeine to digitalis as a heart-tonic in these cases; See urges its use in the same condition, and the author has found it to be as excellent a remedy as its advocates assert. It must, however, be given in sufficient doses, from five to fifteen grains *ter in die.* The efficacy of opium, or, better, small doses of morphine, as a tonic of the heart, is too little understood, especially in the form of hypodermatic injection. Inhalations of oxygen-gas, the internal use of cod-liver oil, and general faradization of the muscles, are expedients of high utility. Nitro-glycerin is a remedy of the greatest value in these cases, or when the heart is weak from any cause. By lowering the peripheral tension of the vascular system, it lessens the work of the heart, and thus obviates a serious danger. By dilating the arterioles of the muscular substance of the heart, it promotes the nutrition of the organ, and in this consists one of the most valuable results of its administration. It relieves the sense of precordial distress, the faintness and vertigo, and other unpleasant consequences of the cerebral anæmia. When the blood-pressure is low because of a relaxed state of the arterioles, barium chloride (gr. $\frac{1}{4}$—gr. $\frac{1}{2}$) is the most efficient of the remedies now in use.

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* J. Lockhart Clarke, "St. George's Hospital Reports," vol. iv, p. 1.

RUPTURE OF THE HEART.

Definition.—Under the designation of *rupture of the heart* is meant the so-called spontaneous rupture, in contradistinction to rupture by wounds and injuries.

Pathogeny and Symptoms.—That rupture shall occur it is necessary that the walls of the heart be weakened by disease. The most frequent cause is fatty degeneration, for, in twenty-four cases, this condition of the muscular tissue was found in nineteen.* Next in importance as a cause is the softening produced by acute myocarditis, especially the suppurative form, or the aneurisms, so called, due to the changes of chronic myocarditis. Diseases of the coronary artery, tumors, echinococci, by destroying muscular tissue, lead to rupture. It is more common in men than in women, and in old age—after sixty years. As to the site of the rupture, statistics show that the left ventricle, at or near the apex, next the right ventricle, then the right auricle, are the most usual; but the preponderance is immensely on the side of the left ventricle—forty-three times in fifty-five cases. There is usually but a single rent, but there may be several, and, as they follow the direction of the muscular bands and the line of least resistance, they are tortuous, somewhat jagged in their margins, and the two orifices are not opposite. The size of the rent varies from an inch to the whole length of the cavity. The pericardial sac contains more or less blood, according to the size of the opening. The rupture may be gradual, a part yielding at a time. Death may take place almost instantaneously. Usually, a groan or a cry is uttered, the face grows deadly pale, the individual falls unconscious, there is some shuddering, and he is dead. The dying may extend over several days—the patient experiencing the symptoms of *angina pectoris* several times with intervals of partial relief, death occurring suddenly at last. In such cases, it is assumed that successive portions of the heart-wall yield, or that clots temporarily obstruct the rent.

The treatment, when there is time for it, is purely symptomatic.

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* "Berliner klinische Wochenschrift," 1873, p. 15; Ponfick, "Ueber Fettherz."

HYPERTROPHY AND DILATATION OF THE HEART.

Definition.—By hypertrophy of the heart is meant an increase of size of the organ, because of an addition to its substance. This enlargement takes several directions, as follows :

Simple hypertrophy means an increase in size without alteration of the cavities ; *concentric hypertrophy* means increase in thickness of the walls, the cavities becoming smaller ; *excentric hypertrophy* means increase in the thickness of the walls, the cavities becoming larger.

The dilatations of the heart correspond in arrangement as follows :

In *simple dilatation*, the cavities are enlarged while the walls remain normal ; in *active dilatation*, which corresponds to excentric hypertrophy, the cavities are enlarged, and the walls are increased in thickness ; in *passive dilatation*, the cavities are enlarged and the walls are thinner. This is the most usual form.

The conditions attendant on hypertrophy and dilatation are, in some respects, the same, so that it is an economy of space, and contributes to clearness of conception, to study them together.

Causes.—*Hypertrophy.*—Simple hypertrophy, which is by no means common, arises from over-action of the cardiac muscle, without there being any disease of the circulatory apparatus. The over-action is due to the abuse of such stimulants as coffee, tea, tobacco ; to moral emotions and intellectual effort, when excessive ; to repeated muscular fatigue, etc. The hypertrophy resulting in this way is general. Any obstacle to the free circulation of the blood imposes additional work on the heart. Narrowing of the aortic orifice gives the left ventricle more work to do, and hence its muscular fibers undergo hypertrophy ; in the same way, hypertrophy of the right ventricle results from narrowing of the pulmonary orifice, of the left auricle, from mitral stenosis, and of the right auricle, from tricuspid stenosis. These are typical examples of partial hypertrophy. The causes of obstruction in front, inducing hypertrophy of the left ventricle, are several : stenosis and regurgitation at the orifice of the aorta ; narrowing of the artery at the duct of Botal ; aneurism, and compression of the vessel by tumors ; atheroma of the arterial system. Hypertrophy of the left auricle results from obstruction and regurgitation at the mitral orifice, especially narrowing of the orifice. Similar causes produce similar effects on the other side. Hypertrophy of the right ventricle is due to narrowing of the pulmonary orifice, to aneurisms, and tumors compressing the artery, to chronic pulmonary diseases which obstruct the circulation, as emphysema, caseous pneumonia, fibroid lung, large pleural accumulations, etc. Hypertrophy results from, or is an attendant on, Bright's disease. Various explanations have been offered of the nature of this relationship, but it is clear that, if hypertrophy of the muscular layer of the arterioles exist in front, the heart has increased resistance, which requires additional effort to overcome. Hypertrophy is, so to speak, a physiological result of the changes in the arterial system due to age; for the calcareous deposition in the tunics of the aorta and of the vessels generally greatly increases the resistance of the arterial circuit by diminishing the elasticity.

Dilatation.—Simple dilatation of the heart occurs in delicate constitutions, especially of growing youths, subjected to over-exertion. This has been observed in armies on a large scale, and by civil physicians as well.* Maclean † has published observations on this point made in the English service; Seits and others in Germany; but Da Costa was the first to set the subject in its true light, by studies in our hospitals during the late rebellion, and preceded all other investigators in this line.

The right ventricle, being much feebler than the left, is more liable to suffer dilatation. This condition results from the increase of pressure due to insufficiency of the semilunar and tricuspid valves, and pulmonary lesions which hinder the circulation in the pulmonary capillaries, such as emphysema, chronic bronchial catarrh, chronic interstitial pneumonia, and tubercular and caseous infiltration. On the left side the most frequent cause is aortic obstruction and insufficiency; but obstruction rather than insufficiency is more certain to produce the dilatation. Mitral insufficiency leads to dilatation of the right cavities by maintaining constantly an increased pressure in the pulmonary capillaries. The cavities yield under normal pressure of the blood when altered by disease. Pericarditis and endocarditis affect the condition of the muscular tissue, by setting up a myocarditis—a granular degeneration. Myocarditis arises under other circumstances also, and the heart-muscle is weakened, not by this disease only, but by fatty degeneration, fatty substitution, tumors, etc.

Pathological Anatomy.—In hypertrophy the change may be confined to one part, or the whole organ may be involved. To such enormous proportions does the heart attain sometimes as to be called *cor bovinum*—ox's heart. The walls of the left ventricle may increase to an inch, an inch and a half, or even two inches in thickness, and the walls of the other cavities undergo corresponding development. The shape of the heart is altered by hypertrophy. When there is hypertrophy of the right ventricle, the heart is widened transversely and the apex is blunted; when the left ventricle is enlarged, the heart is elongated, and, if its cavity is at the same time enlarged, the septum is pressed over into the right ventricle. When both ventricles are enlarged, the heart assumes a globular shape. The position of the hypertrophied heart is more horizontal; if the left ventricle is the seat of the change, the direction of the organ is to the left and downward. By reason of an increase in weight the heart in the recumbent posture sinks relatively lower, and hence the area of absolute dullness may appear smaller; in the vertical position the heart descends, pushing the diaphragm before it, and making the epigastrium more prominent. In texture, the substance of the heart is firmer than normal, and when divided has sharp edges which remain apart. In color, the tint is brighter and fresher looking than in the healthy state. Subsequently, if fatty change begins in isolated patches, the reddish-brown hue of the muscle will be marked by spots of a faintly yellowish or reddish-yellow color. It seems to be well established that the increase in the muscular tissue of the heart is a true hypertrophy, and not a hyperplasia, that the existing elements are increased in size, but that no new elements are formed. Dilatation occurs chiefly in the auricles, which may be so stretched that the muscular elements undergo fatty degeneration, are absorbed and disappear, leaving the endo- and pericardium in contact, or separated by some connective tissue only. The size to which the auricles may be expanded is enormous. The right ventricle may be much dilated and its walls thinned ; the orifices may be much enlarged, the trabeculæ wasted, and the valves thinned. The left ventricle is rarely dilated merely, but the walls are also hypertrophied.

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* Dr. O. Fräntzel, "Ueber die Entstehung von Hypertrophie und Dilatation der Herzventrikel durch Kriegsstrapezen," Virchow's "Archiv," Band lvii, S. 215.

† "The British Medical Journal," February 16, 1867.

Symptoms—*Hypertrophy.*—The signs and symptoms of cardiac disease are divisible into two groups—*rational*, *physical*. The rational signs are presumptive, and consist of the functional disturbances which indicate the probable seat of the disease ; the physical signs are derived from physical laws and methods, and are positive in their results. As respects the rational symptoms, the first point to be noted is, that those vessels receiving their blood-supply from an hypertrophied ventricle obtain more blood and with greater force than in the normal condition, and hence the tension in these vessels is higher ; whereas, the vessels on the other side receive less blood with diminished force, and their tension is lower. When the left ventricle is hypertrophied, the tension is increased in the aortic system and diminished in the pulmonary. The opposite condition obtains when the right ventricle is enlarged, for then the pressure is greater relatively in the pulmonary system and less in the aortic. When both ventricles have undergone hypertrophy, the tension is increased in the aortic system and in the pulmonary artery. In consequence of the increased distributing power of the left ventricle, the blood-current is accelerated in the arterial system and communicating capillaries, and, as the pulmonary circuit has also a higher tension and greater celerity, the blood received from the great venous trunks is quickly disposed of, so that the tension falls in the venous radicles. The final effect of pure hypertrophy is an acceleration in the whole round of the circulation. The pulse is full, firm, and bounding. The ascent line of the sphygmographic trace is

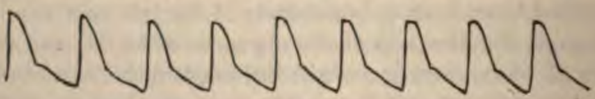

FIG. 22.—Hypertrophy.

vertical and abrupt, but the summit is rounded and the descent oblique, unless there be regurgitation at the aortic orifice. The face is red and congested; the nose bleeds easily; the head feels full, and aches a good deal, especially when any strong muscular effort is made; there are more or less *tinnitus aurium* and dizziness. When the arterial walls are weakened by atheromatous degeneration, cerebral hæmorrhage may be a result of hypertrophy of the left ventricle; but the way to rupture is prepared by gradual yielding of the arterial tunics, and the formation of minute aneurismal dilatations known as "miliary aneurisms." The strong beating in the superficial arteries is felt by the patient, and produces a disagreeable roaring and beating in the ears, especially when lying on the left side. The attacks of palpitation are frequent, but their severity is not in proportion to the extent of the hypertrophy, for the action may be very tumultuous when the enlargement is slight, and *vice versa.* There are pretty constantly felt by the patient a sense of præcordial anxiety, and, rarely, attacks of pain extending to the shoulder and arm, similar to *angina pectoris.* A sense of fullness in the chest, of oppression, and sometimes embarrassed breathing are experienced, but the pulmonary symptoms may be due to congestion of the bronchial mucous membrane, supplied as it is by the bronchial arteries, and not from the pulmonary. When the hypertrophy is confined to the right ventricle, no other lesion existing—an extremely rare condition—the symptoms present will be a sensation of fullness and oppression of the chest—possibly dyspnœa; œdema and hæmorrhage may occur, and the production of interstitial inflammation and possibly other diseases promoted. The foregoing signs of hypertrophy are presumptive or rational; the physical signs now to be considered establish the seat and character of the lesion. On *inspection* there is to be observed a prominence of the chest, greatest at the junction of the fourth and fifth ribs with the sternum. This has been denied; but, that it is often encountered in hypertrophy occurring in young subjects, the author's experience entitles him to affirm. When hypertrophy occurs later in life, the ribs having become rigid, no elevation of the chest-wall can be effected, how powerful soever may be the impulse of the heart. As in hypertrophy, the position of the heart is more horizontal and depressed to the left, on *palpation*, the apical impulse is felt near to the axillary line, and one, two, and possibly three intercostal spaces lower down, and it is stronger and more widely diffused. The force of the impulse is sufficient to raise the hand when placed on the cardiac region, or the head when applied in auscultation, and the whole left thorax may be felt lifted up and carried toward the left. This is entitled *the heaving impulse,* and is very characteristic of extreme hypertrophy. Instead of the impulse having a heaving character, sometimes it makes the impression of a sudden jar which is immediately arrested. In hypertrophy of the right ventricle the heaving impulse is felt at the end of the sternum, especially its right border, and in the epigastrium. In the third and fourth intercostal spaces to the right of the sternum, the impulsion of the hypertrophied auricles may sometimes be felt. On *percussion*, the area of præcordial dullness can be demonstrated. The *absolute* or superficial dullness is that derived by percussion over that portion of the heart uncovered by the lung—a triangular space; the *relative* or deep dullness is that obtained by strong percussion over that portion of the heart covered by the lung. The dull space extends from a point internal to the upper border of the second rib at its junction with the sternum, obliquely downward to the left to the apex-beat, thence transversely to the right border of the sternum. This is an irregularly triangular or ovoidal space which returns, on percussion, the forms of dullness mentioned above. The area of absolute dullness is increased by hypertrophy of the heart, if the patient is percussed when erect and inclined slightly forward. The relative dullness is increased more when the patient is recumbent, by the heart sinking backward. In hypertrophy of the left ventricle, the dullness is parallel to the long axis of the heart; in hypertrophy of the right, the dullness is over the lower extremity of the sternum.

When pure hypertrophy is the condition under examination, *auscultation* furnishes no important information. The sounds of the heart are somewhat affected in their timbre. In hypertrophy of the left ventricle, the first or ventricular sound has a rather metallic quality, and the second sound is strongly "accentuated"; in hypertrophy of the right, the same facts exist, but the sounds are less intense. At the apex, a peculiar metallic "click" is sometimes heard, and is doubtless due to the vibration in the chest-well, produced by a very strong impulse. It is much louder when the stomach is distended with gas.

Dilatation.—When dilatation occurs in any of its forms, the propulsive power of the heart is diminished; less so, however, in *active dilatation*. The result of this is a condition of ischæmia in one set of vessels, and of stasis in the other system. Thus, when the left ventricle is dilated, there is a lowering of tension in the aortic system, and an increase of pressure and abnormal fullness of the pulmonary; when the right ventricle is dilated, there are diminution of tension, and ischæmia of the pulmonary artery, and elevation of pressure with stasis in the peripheral venous system. The ultimate effects of the disturbance in the vascular system are the same when one ventricle is dilated as if both were, for, taking as an example the most common dilatation, that of the right side of the heart, the stasis in the peripheral veins extends to the capillaries, to the arteries, thence to the left side, and *vice versa*. When, however, dilatation of the right ventricle coincides with hypertrophy of the left, the excess in power of the one compensates for the deficiency in the contractile energy of the other. The results of dilatation of all the cavities are these: the vessels receiving blood from the heart—efferent vessels—are in a condition of ischæmia, or diminished blood-supply, while the vessels conveying the blood to the heart —afferent vessels—are constantly abnormally full, or in a condition of hyperæmia and exaggerated tension. When the right heart is dilated, there are ischæmia of the pulmonary vessels, producing habitual dyspnœa, insufficient hæmatosis or aëration of the blood, and stasis in the general venous system. The peripheral veins are turgid with blood, there is cyanosis from deficient aëration, and a constant hyperæmia of the liver, spleen, kidneys, and intestinal canal. Increase of pressure in the renal veins causes *albuminuria;* in the hepatic veins, jaundice and ascites ; in the veins of the extremities, œdema and general dropsy, and thrombosis. The rational symptoms of these functional disturbances are, palpitations of the heart ; frequency and irregularity of the pulse ; deficiency in the arterial blood-supply to the brain, and manifest in vertigo, ringing in the ears, attacks of faintness or actual syncope, etc. ; deficiency in the blood going to the lungs, and causing cough, dyspnœa, etc. The composition of the blood is impaired by the excess of carbonic acid ; the lessening of the oxidation processes diminishes the production of heat, and hence the general temperature is low ; the vessels themselves, the heart, and the tissues, undergo nutritive changes in consequence of insufficient energy in the process of tissue metamorphosis. A cachectic state, with lowered vitality of the tissues, so that they ulcerate under the least irritation, is the necessary outcome of these changes. There is not only a lowered state of the assimilative functions, but elimination is imperfectly carried on, and excrementitious materials are retained in the blood—carbonic acid and urea—causing hallucinations, delirium, eclampsia, coma, etc. The ill results of these nutritive alterations are also exhibited in increased damage to the heart-muscle, and consequently an exaggeration of the mechanical effects of the dilatation. Inspection furnishes no information of value, except, when dilatation of the right cavities render the valves incompetent, a venous pulse will be visible in the neck. On palpation, the area of cardiac impulsion is as wide as in hypertrophy, but the apical impulse is feeble, and may not be felt when the patient is recumbent. When there is hypertrophy of the right heart to compensate for dilatation of the left cavities, the apical impulse will be feeble, while the pulse of the right cavities at the border of the lower sternum will be comparatively strong. On percussion the extent of dullness is made out as in hypertrophy. On auscultation, the sounds are feeble, as a rule ; on the other hand, they may have a more clear and resonant quality. A soft-blowing murmur sometimes takes the place of the first sound. This murmur is situated in the mitral and tricuspid areas, and is due to the insufficiency of the valves to close the auriculo-ventricular orifices.

Diagnosis.—Hypertrophy is to be distinguished from dilatation of the heart, from pericardial effusions, tumors of the mediastinum, etc. The force of the impulse, the accentuation of the second sound, and the state of the systemic circulation, enable the differentiation to be made from dilatation, and also from effusion ; besides, in the latter, the dullness has been preceded by a friction-sound, and, when the effusion comes on, the heart-sounds weaken and disappear. The seat of the dilatation is determined chiefly by the position of the dullness. Hypertrophy and dilatation are differentiated from tumors in the mediastinum, by the displacement of the heart occasioned by the latter, and by the persistence of the normal heart-sounds. The pressure of a tumor on the great vessels and important nerves introduces into the symptomatology of the case new symptoms quite foreign to either hypertrophy or dilatation. From pleuritic effusion in the neighborhood, retained by adhesions—the so-called encapsulated—the dullness due to hypertrophy or dilatation may be difficult to separate, but effusions displace the heart without altering the character of its impulse and its murmurs ; when the pleural effusions are unconfined, the ready distinction consists in the change of the position of the patient, shifting the dullness.

Course, Duration, and Termination.—The course of these affections is chronic, but hypertrophy continues much longer than dilatation. Hypertrophy, uncomplicated, exists unchanged for many years, and is important rather on account of the complications which may grow out of it than of itself, yet changes in the heart-substance and in the vessels must eventually result. Over-supply of blood to organs leads to nutritive alterations in them. Rupture of vessels may take place, but disease of the arterial tunics is necessary also ; hence the importance of hypertrophy of the heart as a factor in cerebral and in pulmonary hæmorrhage. Dilatation of the cavities is much more rapid in its course and important in its results than hypertrophy, but simple and passive dilatations are more serious than the active form. The heart is much weaker, its tissues become diseased, and death may be sudden by paralysis or by rupture, or in attacks similar to angina pectoris. The stasis in the circulation, the pulmonary, hepatic, and renal troubles, and the general dropsy which result from dilatation, are the usual sequelæ, and death ultimately occurs from the combined effect of these disturbances.

Prognosis.—The prognosis is necessarily grave, but it should always be guarded. Simple hypertrophy may exist for years, without any apparent interference with function. In dilatation, the hope of any lengthened period of freedom from ill results can not be encouraged. When dropsy appears, it becomes a question of the physical endurance largely, for death can not, then, long be delayed.

Treatment—*Hypertrophy.*—When hypertrophy is compensatory or compensated, there is no need of therapeutical measures. It may, however, be necessary to combat the hypertrophy, or its results in the organism at large, if the force of the heart and the pressure in the vascular system are so great as to threaten serious consequences. The most direct method is the abstraction of blood, either by venesection or by leeches, and this is allowable in vigorous subjects. Purgatives lower the blood-pressure, especially the saline purgatives, which draw off by the intestinal mucous membrane more or less fluid. They are much less objectionable than bloodletting, are more easily handled, and are more permanent in results. Next to saline purgatives in efficiency is the tincture of aconite-root. Tincture of veratrum viride is more powerful, but less easily managed, for its effects are quickly produced and not easily confined within the prescribed limits.

The treatment of dilatation must pursue the opposite direction. The general nutrition must be maintained at the highest point, to promote the nutrition of the cardiac muscle. A generous diet, moderate exercise in the open air, the inhalation of oxygen, are important agencies to accomplish the objects just mentioned. Bitters to increase the appetite and iron to improve the quality of the blood are strongly indicated. To tone up the heart and raise the tension of the vascular system, there is no remedy so efficient as digitalis. It should be given with quinine, which is also an excellent heart-tonic. The most remarkable effects attend the use of minute doses of morphine hypodermatically in these cases. When there is extreme dyspnœa, the heart very feeble, the fluid everywhere gaining, the effect of the injection is almost magical. It sometimes happens that the symptoms are too urgent to await the slow action of digitalis, or it may be the stomach will not tolerate the digitalis in any form, then the injection is most opportune—the patient is relieved by it—time is gained for the action of the various remedies that may be substituted for it, or the stomach will bear it better. A decided change has taken place in the state of professional opinion on the subject of exercise in heart-diseases. From absolute quiet, or the avoidance of anxiety and fatigue, opinion has gone in some extreme instances to mountain climbing. It need hardly be said that the medium is the true ground to take. Daily, systematic, walking exercise, such as will improve the forces of the body, contributes to improve the heart nutrition and the increased muscular action its muscular power.

Careful attention should be given to the diet of these patients. In many instances acidity of stomach causes reflex palpitations of a very hurtful kind, and any articles difficult of digestion, especially if nausea follows, is apt to excite paroxysms of pseudo angina pectoris.

ENDOCARDITIS—INFLAMMATION OF THE ENDOCARDIUM.

Definition.—Acute inflammation occurs in two distinct forms: plastic, or simple exudative inflammation; ulcerous, or diphtheritic inflammation. The plastic form is either acute or chronic, but these differ merely in degree and rate of progress.

Causes.—Primary or idiopathic endocarditis, except in the ulcerous form, is extremely rare. Plastic endocarditis is usually a secondary affection : secondary to pleuritis, pneumonia, pericarditis, myocarditis, etc., but, very much more frequently, secondary to acute rheumatism. The relative frequency of endocardial inflammation in acute rheumatism is differently stated by different observers. According to some, one half, others one third, of the cases are complicated by endocarditis, but the real number is, no doubt, lower than one third. The source of error is the occurrence of a soft-blowing murmur in cases of rheumatism, due not to inflammation of the endocardium but to the condition of the blood. The more severe the type of rheumatic fever the greater the danger of cardiac complications, but there are numerous exceptions to this rule. The pericardial and endocardial inflammation may precede the joint-troubles.

Pathological Anatomy.—The initial lesion is hyperæmia, which involves the sub-serous connective tissue as well as the membrane itself. The stasis in the vessels induces rupture of the capillaries, here and there, and minute extravasations are thus formed. Migration of white corpuscles, exudation of fibrinogenous and germinal matter, now takes place into the affected membrane, and the cells of the endothelium become cloudy, loosen, and undergo proliferation. The membrane, which in health is thin, transparent, and glistening, becomes, as a result of these changes, rough, opaque, and thickened. The roughness of the membrane is due, further, to the formation of lamelliform or conical vegetations, the product of the activity in cell proliferation at particular parts, or, according to Rindfleisch, they are composed of an homogeneous fibrinous exudation from the vessels. If the changes in the structure of the membrane do not go beyond this point, it is probable that complete restitution may occur. Proceeding from this point the inflammation may take the *plastic* or the *ulcerous form.* We are now concerned with the former only. The exudation on the auriculo-ventricular valves (mitral) is found chiefly at the free border, where the tendons are inserted ; on the semi-lunar valves (aortic) on the lateral border where the segments come in contact, yet the corpora arantii may also be the seat of abundant exudation. The vegetations projecting from the surface of the membrane entangle masses of fibrin whipped out of the blood, which may project from the valves, swinging to and fro like a polypoid excrescence. The chordæ tendinæ may be affected in a manner similar to the valves. Softened by the inflammatory process, the chordæ may give way, permitting a segment to become adherent to a neighboring one. Adhesion of the semi-lunar valves may occur at the side where they are in contact. The adhesions undergo organization, and thus the most serious changes are wrought in the structure and functions of the valves. Also, large masses of fibrin may be entangled in them, and they may be the cause of thrombotic deposits around them. When the inflammatory process passes to the chronic stage, characteristic changes take place in the exudation : it loses some part of its water, solidifies, and subsequently contracts. The connective tissue undergoes hyperplasia, especially the connective tissue of the borders of the valves, but the membrane, generally of the valves, may be affected by the same change. As a result of the tendency of the new material to contract, the valves become much deformed, thick, and inflexible, and, of course, their functions are correspondingly impaired. Calcareous changes occur in the deposits, and fatty degeneration also takes place. Patches of softening may occur in the valves, the membrane yields, and pouches or aneurisms form, which ultimately give way, and thus a valve is perforated. This process, occurring at various points, imparts to the valve a sieve-like appearance. Vegetations detached, or bits of adherent fibrin cast off, constitute emboli, which, entering the blood-current, will be deposited in distant parts—on the left side of the brain, in the kidneys, spleen, etc. The orifices of the valves undergo similar changes. The connective-tissue transformations take place, and hence rigidity, deformities, and contraction result.

Symptoms.—When endocarditis is idiopathic, which is very rare, its onset is marked by the usual symptoms of an acute febrile or inflammatory affection. There is a chill, followed by fever, a coated tongue, anorexia, nausea, sometimes vomiting, and general *malaise.* As it occurs in the course of another disease, the additional disturbance induced by it may altogether escape recognition, and it is only by persistent watchfulness, under such circumstances, that it is discovered. This is true of its onset in rheumatism, Bright's disease, the eruptive fevers, etc. On the other hand, the commencement of endocarditis may be manifest by very obvious signs. For example, if during the course of acute rheumatism endocarditis comes on, there will occur an increase in the temperature, the thermometer rising a degree or two, the pulse will become more rapid, and the general condition less favorable, than before the complication arose. The fever does not pursue a special type, and the pulse exhibits no characteristic quality. The other rational symptoms are equally indefinite. There may or may not be some uneasiness in the region of the heart, some præcordial oppression, and some palpitation. There may occur, also, increased impulsion of the heart, more rapid and tumultuous beating of the carotids, headache, noises in the ears, some dyspnœa, etc. After a time the action of the heart becomes less energetic, the strength of the pulse declines, the function of hæmatosis is impaired, and hence the functions generally, especially the cerebral, are less energetically performed. The physical signs are much more distinctive than the rational; the changes in the valves and at the orifices necessarily modify the character of the murmurs, or add new sounds. The period and position of the murmur are determined by the valve affected and by the time, in the cardiac revolution, when the blood-current passes the affected orifice. In mitral insufficiency a *bruit* or murmur is audible with the first sound (systolic) at the apex, and with the second sound (diastolic), or after it (presystolic), if there is obstruction at the mitral orifice. In aortic obstruction the murmur is audible with the first sound (systolic) at the base, and with the second sound (diastolic) if the aortic valves are insufficient. If the lesions occur on the opposite or right side of the heart, which is very rare, the same rules obtain, but the position at which the sounds are heard is different. To hear the sounds at the right auriculo-ventricular orifice, the ear must be placed over the ensiform appendix, and, for the pulmonary valves, at the junction of the third right rib with the sternum. Percussion affords but little information. If there be aortic obstruction, some distention of the heart is occasioned, which increases the area of dullness in the vertical direction; if mitral obstruction, the right cavities will be somewhat dilated and the dullness increased in the transverse direction. The facts may be formulated as follows: In acute endocarditis the same physical signs characteristic of chronic valvular diseases of the heart occur suddenly; and, further, the sudden development of the symptoms of mitral insufficiency is the most characteristic sign of acute endocarditis (Jaccoud). Obstruction or regurgitation at the mitral orifice increases the pressure of the blood in the pulmonary artery, and hence a physical sign of this condition is accentuation of the pulmonary second sound. More or less congestion of the lungs and stasis in the venous system are necessary consequences of mitral disease.

Course, Duration, and Termination.—The course of acute plastic endocarditis is necessarily brief. The patient either partially recovers by the disease assuming the subacute and chronic phase, or he dies from the immediate consequences and complications. When the case passes from acute to chronic, the fever ceases, compensation takes place, by which the disorders of circulation are obviated for a time, yet the physical signs of valvular mischief continue. Death may result from a gradual weakening, terminating in paralysis of the heart, or heart-clot may form, or a cerebral embolism occur. Pericarditis, myocarditis, and pneumonia, may also intervene and take life. That a cure of actual lesions may happen is admitted, but the examples of such a fortunate termination are extremely infrequent. The duration of the acute attack is short; of the subacute and chronic form, indefinite.

Diagnosis.—The differentiation consists in the application of the physical signs. It should not be forgotten that a murmur exists of a soft-blowing character, not due to valvular lesion, and which disappears on the subsidence of the acute symptoms.

Prognosis.—The acute form is not very dangerous to life, and hence a favorable prognosis may be expressed. As regards the ultimate results of valvular lesions, the prognosis is grave.

Treatment.—The character of the associated malady and the condition of the patient must enter largely into the consideration of remedies. As it is a fundamental principle to keep the suffering organ quiet, remedies capable of effecting this are very important—these are, ice and digitalis. An ice-bag should be applied to the præcordial region, and a tablespoonful of infusion of digitalis given every four hours. Flying-blisters should be applied to the axillary region. In the incipiency, before much damage has been done, there can be no doubt of the great efficacy of the hypodermatic injection of morphia, or the internal administration of morphia and quinine—one quarter grain of morphia and ten grains of quinia every four hours until three or four doses are taken. When considerable exudation has occurred, besides the remedies to quiet the heart, ammonia should be given freely, with the view to exert a solvent action. The best form for administration is the carbonate (ten grains) in the solution of the acetate (half an ounce) every four hours, or half the quantity every two hours. If there be much depression in the progress of the case, quinine and digitalis should be prescribed in combination, or caffeine, strophantine, chloride of barium, and other remedies of the kind should be given.

ULCERATIVE ENDOCARDITIS—DIPHTHERITIC ENDOCARDITIS.

Definition.—This is a peculiar form of disease, in which ulcerations and diphtheritic exudations, with colonies of micrococci, develop in the endocardium, followed by septic infection of the blood and multiple embolisms.

Causes.—A peculiar state or type of constitution seems necessary to develop this disease. It occurs during the course of some cases of acute rheumatism, of puerperal fever, of diphtheria, etc., and now and then this process attacks the valves in cases of chronic plastic endocarditis, the new material undergoing rapid and destructive ulceration. This disease makes its appearance between puberty and forty. A depressed condition of the vital forces, due to bad hygienic influences, seems to be very influential in determining the occurrence of this disease in youths. The close analogy between the diphtheritic process and this ulcerous disease of the left heart and the frequent coincidence of the two affections render it highly probable that the diphtheritic poison is the chief if not the only factor in its causation.

Pathological Anatomy.—The initial lesions are the same as those described under the head of plastic endocarditis. The lesions are chiefly on the left side of the heart, and attack by preference the anterior flap of the mitral and the semi-lunar valves of the aorta; next the walls of the appendages to the left auricle; and, lastly, the walls of the ventricle. Occasionally the same morbid process occurs on the right side, and, in one reported case, on the tricuspid only,* and its chordæ tendinæ, which were destroyed. After the initial changes already described, the nuclei of the connective tissue undergo rapid proliferation and form granulations of the surface; fibrinous deposits take place, and the whole forms a "felt-like" mass, intimately connected with the tissues beneath. A process of softening then begins in the interior of these masses; they crumble and fall away, and leave a ragged, irregular ulcer, which is the seat of fresh fibrinous deposits. Perforation of the valve may ultimately take place, and the margins of the perforation are rough, ragged, and ulcerated; and they are surrounded by granulations having the same structure as those which have already ulcerated. A distinctive peculiarity of this process is the presence early in the course of formation of the granulations, and in the midst of the proliferating connective-tissue corpuscles, of a finely granular material, the particles having various shapes, strongly refractive of light, and resisting the action of acids and alkalies. These granules, as Virchow was the first to point out, are micrococci, and the granular masses are colonies of micrococci. The losses of substance by thinning the valves lead to the formation of the so-called valvular aneurisms, and coagula forming in these are thrown off with patches of diseased tissue, when the aneurism gives way. Ulceration of the septum, induced in the same way, leads to communication between the cavities. The particles of ulcerating tissue, of fibrin and blood-clot, and the little masses of micrococci colonies thrown off into the blood-current, form multiple embolisms. Two results follow: either there is merely mechanical obstruction of vessels, or an infective process is set up the same as that of the original disease. The spleen, kidneys, and brain, are the organs in which these deposits take place from the left side of the heart. When the disease is in the right side of the heart, the emboli are swept into the lungs.† As these organs contain the "terminal arteries" of Cohnheim, there will occur hæmorrhagic infarctions and ichorous suppuration. All the organs of the body may, indeed, be the seat of abscesses from embolic deposits. The distribution of infective materials—specific micrococci—sets up a general infection of the blood. Wherever the micrococci are deposited they undergo rapid multiplication, and initiate the same morbid action as at the original source of infection. Numerous are the alterations occurring in various organs in ulcerative endocarditis. The spleen is very much enlarged, whether the seat of infarctions or not; in the kidneys are abscess formations, and the afferent vessels are blocked with colonies of migrating micrococci; in the brain there are extravasations, especially of the meninges; in the lungs, abscesses from emboli; in the heart, myocarditis and pericarditis; and in the small intestine, swelling of the patches of Peyer and solitary glands, and ulcerations which differ from those of typhoid, in that they are not confined to the lower extremity of the ilium, are not opposite the insertion of the mesentery, and are not limited to the glands.*

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* T. Whipham, M. B., "Transactions of the Pathological Society," vol. xxii, p. 118.

† C. J. Eberth, Virchow's "Archiv," Band lvii, "Ueber diphtherische Endocarditis."

Symptoms.—Cases of ulcerative endocarditis differ much in their objective symptoms, but they may be referred to two types: typhoid; pyæmic. In both, the cardiac symptoms are quite masked by the preponderating importance of the systemic state, and hence cases of primary endocarditis are apt to be overlooked. When there is an attack of rheumatism going on, suspicion of cardiac mischief will of course be excited by the sudden occurrence of a violent chill which inaugurates both forms. In the *typhoid form* succeeding the chill there is considerable fever, the range of temperature being rather of the remittent type; headache, vertigo, and extreme prostration, and sometimes a sense of præcordial oppression, are then experienced; the tongue is dry and brownish; there are nausea and vomiting, and the bowels are constipated, or diarrhœa is present. The prostration gains rapidly, and by the fourth day a condition of depression is reached comparable to the second week of typhoid. The resemblance to typhoid is all the greater, since the abdomen is swollen and tympanitic and the spleen is enlarged. Delirium (irritation) soon comes on, to be replaced in a few days by stupor and coma (depression). A severe diarrhœa now succeeds to constipation, if that condition has existed before, and the perplexity of the case may be enhanced by rose-spots and petechiæ appearing on the abdomen. Presently, the patient lying in a comatose state, the stools and urine are passed involuntarily. The urine has a smoky appearance, and contains more or less blood, and albumen is present. There is usually some bronchial catarrh, with cough and dyspnœa—the latter, however, may be due to blocking of vessels and infarctions. On auscultation, a rather loud, systolic murmer is audible, usually with greatest intensity in the mitral area, or with the second sound in the aortic area. The *pyæmic* form begins with a chill, which is a decided rigor, followed by a high fever and sweating. The chills recur sometimes with the regularity of an intermittent fever, but usually very irregularly, as is proper to pyæmia. A condition of profound and increasing adynamia is soon developed. There is often a yellowish hue of the skin; there may be jaundice, or there may occur petechial or hæmorrhagic spots, or a roseola may make its appearance. During the maxima of the temperature curves the heat may attain to 105° Fahr. and the pulse to 140. Dyspnœa and accelerated breathing may indicate pulmonary infarctions and pneumonia; enlargement of the spleen (infarctions of that organ); renal pains, albuminuria and hæmaturia (infarctions of the kidneys); and apoplectic attacks and hemiplegia (infarctions of the brain). Abscesses occur in the joints in a considerable proportion of cases. They are peculiar, in that they form with great rapidity; are, when at rest, free from pain; and are not manifest by swelling and changes in the form and appearance of the joint. In some cases there occurs an acute atrophy of the liver, with an intense icterus. Confusion of mind is observed with the onset of the symptoms, then an active delirium, followed in a short time by stupor, coma, and insensibility. Not all the cases conform to one or the other of these types; some pursue an intermediate course; others seem to be only aggravated cases of rheumatic fever. There may be no physical signs to warrant the opinion that endocarditis exists; there may be no marked affection of the joints—only vague pains in them, and in the muscles, yet there are maintained a high grade of temperature and a rapid pulse, and the stomach continues much deranged.

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* Rudolf Maier, Virchow's "Archiv," Band lxii, "Ein Fall von primärer Endocarditis diphtheritica."

Course, Duration, and Termination.—The course of ulcerative endocarditis is very rapid, the pyæmic form being more quickly fatal. This form rarely continues longer than ten days, and many terminate within a week. On the other hand, the typhoid form may last three or four weeks, or even longer. Death may occur from paralysis of the heart, from heart-clot, from thrombus of the pulmonary artery, from pneumonia, from cerebral embolisms, etc.

Diagnosis.—A typical case of the typhoid or pyæmic form, occurring in the course of acute rheumatism, ought to be diagnosticated without difficulty. Generally the symptoms do not indicate the nature of the lesions. Probably ulcerative endocarditis is more frequently confounded with typhoid than any other malady. The differentiation can not be made from the symptoms, but from the history of the case. In typhoid there is slow development, and the grave symptoms do not come on until the first week is passed. The circumstances surrounding the individual and the occurrence of other cases in the neighborhood must be taken into account.

Treatment.—Notwithstanding the apparently hopeless condition of the patient affected with ulcerative endocarditis, our efforts should be directed to the use of stimulants and support, and special remedies, as if there were a prospect of cure. As septic materials are circulating through the blood, the benzoate of ammonium, or salicylic acid, should be administered freely. To effect the solution of blood-clots and fibrin masses, we should keep the blood as highly alkalinized as possible by ammonium carbonate. Quiniæ and morphia are the appropriate remedies during the first few days; carbonate of ammonia and the benzoates, when the endocardium is disintegrating, and alcoholic stimulants and abundant food-supply throughout the whole duration of the case.

DISEASES OF THE VALVES AND OF THE ORIFICES.—VALVULAR LESIONS.

Definition.—Under the term "valvular disease" are included those alterations in the structure of the valves themselves, or of the orifices, which render the former incapable of performing their office in the closure of the latter. The lesions may be of two kinds—*obstructive*, or *regurgitant*; that is, the orifice may be so narrowed as to obstruct the passage of the blood, or the valves may be so damaged as to permit the blood to regurgitate. The narrowing of an orifice is termed *stenosis*; the incompetence of a valve to close the orifice is termed *insufficiency*; as aortic stenosis, mitral insufficiency, etc. There are four points at which these lesions may occur: on the left side, at the auriculo-ventricular orifice (mitral), at the aortic orifice (semi-lunar); on the right side, at the auriculo-ventricular orifice (tricuspid), at the pulmonary orifice (semi-lunar).

Causes.—There seems to be no difference in the liability of the two sexes respectively to the occurrence of valvular diseases. Age exercises a very manifest influence in the production of aortic disease, by the development of atheromatous changes, while mitral lesions occur more frequently in youth. Still, the rule is not invariable. Aortic disease may be brought on in early life by overwork and strain of the heart, as was first pointed out by Da Costa. According to Bamberger, the greatest frequency of mitral disease is from ten to thirty, and of aortic disease from thirty to fifty. The relative proportion of cases fatal from heart-diseases, in the deaths from all causes, is differently stated by different observers, from two per cent. to twenty, but the lowest estimate is probably nearest the truth. The most important cause is, doubtless, rheumatic endocarditis, which affects all the valves, but greatly more frequently the mitral. The next in importance as a factor is chronic endarteritis, or atheromatous degeneration, which usually affects the aortic orifice. Syphilis is also a cause, but the precise value of its influence in lighting up mischief in the valves is not known, and, as gummata are deposited in the walls of the heart, the lesions of the valves are usually secondary to myocarditis. Leared * reports a case *supposed to be syphilitic*, in which vegetations formed on the aortic valves, the patient having had recently a well-marked constitutional syphilis.

Rational Signs and Symptoms of Valvular Defects.—When the normal course of the circulation through the heart is disturbed by changes in the orifices and in the valves, certain consequences ensue to the heart itself, and to the organs in general. When stenosis exists at an orifice, the amount of blood passing through is necessarily lessened, with the effect to cause ischæmia and lowered tension in front, and stasis and abnormally high tension behind. The same result follows if the contractions are feeble and the cavity dilated, for then the amount delivered in front is lessened, and accumulation takes place behind. Lesions of the aortic orifice, either obstructive or regurgitant, lead to dilatation of the left ventricle, to diminished blood-supply, and lowered tension in the vessels of the aortic system, and to increased pressure and distention in the left auricle and pulmonary veins. Mitral lesions, either obstructive or regurgitant, cause abnormal fullness and distention of the left auricle and pulmonary system, and ischæmia and lowered tension in the left ventricle and aortic system. Again, lesions of the tricuspid orifice induce dilatation of the right auricle and increased pressure in the venæ cavæ, and ischæmia and lowered pressure in the right ventricle and pulmonary artery. Also, lesions of the pulmonary orifice bring about dilatation of the right ventricle, and elevated tension in the right auricle and venæ cavæ, and ischæmia and lowered tension in the pulmonary artery. Although obstruction and regurgitation of the aortic orifice affect first the aortic system, yet ultimately the dilatation of the left ventricle, and the changes in the auriculo-ventricular orifice will lead to incompetence in the mitral and general venous stasis. The same fact is true of mitral stenosis and regurgitation: the arterial system does not receive its normal supply, and accumulation takes place in the pulmonary veins, and next in the right cavities. Obstruction and regurgitation on the side of the right heart lead to ischæmia in the pulmonary artery, then of the pulmonary veins, then of the left cavities, and finally of the aortic system, while stasis and high tension obtain in the venous system. The final result of valvular lesions on the circulatory system may be formulated as follows: All valvular lesions bring about, sooner or later, a state of the circulatory organs in which there are ischæmia and lowered tension in the aortic system and stasis and higher tension in the venous system. When compensation takes place, this formulated expression ceases to be applicable. By the term *compensation* is meant an adaptation of the organs of circulation to the new conditions imposed on them by the valvular lesions. Stenosis of an outlet is compensated by dilatation of the cavity and hypertrophy of the walls. Thus, in aortic stenosis, some dilatation of the cavity enables the heart to retain the excess in the quantity of the blood, and hypertrophy of the walls enables the left ventricle to deliver the whole amount into the aorta. In this way the obstruction is compensated, so that the subjects of aortic stenosis are enabled to live in comparative comfort for many years. But the compensation may be easily ruptured or overcome. Any unusual work put on the heart, new obstacles introduced by disease in the lungs, or in the heart itself, may disturb the compensatory relation, and the symptoms of valvular disease be resumed again with renewed force.

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* Dr. A. H. Leared, "Aortic Valve-Disease, apparently caused by Syphilis," "Path. Soc. Transactions," vol. xix, p. 94.

The slowing of the current, which is a consequence of stenosis, of changes in the heart-muscle, and of stasis at some point in the circuit, has a disastrous effect by the formation of heart-clots. Coagula form in various situations: on the walls of the heart, entangled in the trabeculæ, or in the auricles. These coagula are found more frequently on the right side, and hence hæmorrhagic infarctions in the lungs are results of valvular disease. A true infarction is possible in those organs only supplied with Cohnheim's terminal arteries. An embolus lodged in one of these stops the blood-current, and, the terminal artery having no anastomoses, there can be no collateral circulation; but in the efferent vein, supplied through a communicating vein by an unobstructed artery, a recurrent movement of the blood takes place, flows on into the capillaries, then finally into the artery with a rhythmical movement. The result is, the wedge-shaped area supplied by the obstructed artery becomes deeply injected, and, vessels yielding under the increased pressure, a hæmorrhage occurs. Thus is produced the pathological state called "hæmorrhagic infarction." If the infarction is large, or if several smaller ones unite, symptoms of disturbance in the pulmonary functions will be induced. There will be dyspnœa, mucous expectoration with more or less blood, chilliness, and the physical signs of consolidation—dullness on percussion and bronchial voice and breath sounds—the latter, however, recognized if the area of infarction be large and situated at or near the periphery. If the pleura is involved there may be pain and fever, but usually the temperature remains rather below than above the normal. In some cases the infarction may be entirely healed, and nothing remain but a cicatrix; in others, if the embolus be infective, a gangrenous inflammation may take place; in others, again, death may occur suddenly from blocking of a considerable vessel.

The most usual pulmonary disturbance induced by valvular disease is stasis of the blood, which leads to catarrh of the bronchi, and is accompanied by cough, by mucous expectoration, mucous and sub-mucous *râles*, etc. Very important changes ensue in the intima of the vessels, and in the caliber of the capillaries; the former undergoes an atrophic change, the latter enlarge and become varicose, and, projecting into the alveoli, narrow the breathing-space, and thus cause dyspnœa. Under the increased pressure, vessels give way and hæmorrhage occurs in the alveoli and intervening connective tissue; and the blood undergoing the usual transformation, produces the so-called "red-brown induration." When the stasis has continued for a long time, and is extreme, the pulmonary tissue becomes œdematous. Difficulty of breathing is a necessary result of these conditions. Besides this habitual difficulty of breathing, there are paroxysmal attacks of considerable severity, in which, without any increase in the number of respiratory movements, there is a sense of need of air, accompanied often by pain in the chest, in the shoulder, and extending down the arm. These attacks are more usual in cases of disease at the aortic ostium, due to atheromatous degeneration. In consequence of the slow circulation through the tissues, the blood loses more oxygen and takes up more carbonic acid ; in consequence of the interference with aëration caused by the pulmonary changes, the blood contains always more carbonic acid and less oxygen than is normal—hence cyanosis is a symptom in these cases. It exists, in varying degree, from a decided blueness of the whole surface to a faint blueness of the lips only. The condition of over-fullness of the venous system is further seen in the distended state of the superficial veins. The increased tension of the veins is an efficient factor in the production of œdema, the absorption of fluid is hindered from the same cause, and the state of the blood-serum favors outward rather than inward osmosis. The accumulation of fluid in the areolar tissue first occurs in the inferior extremities, and then gradually extends upward. Of the internal cavities, the peritoneum becomes earliest and most abundantly the seat of effusion, because of the changes which take place in the liver in these cases of cardiac disease (see CONGESTION OF THE LIVER). Next to the peritoneum, the left pleural cavity contains the most transudation ; next the sac of the pericardium. The severe pressure on the skin of the legs, which is also filled with serum, leads to inflammation of the skin ; it becomes tense, brawny, and congested, and finally ulcerates, forming a more or less extensive purplish excavation, exuding serum constantly. The ulcer or ulcers thus produced are liable to attacks of erysipelatous inflammation, to sloughing, and to deep-seated, burrowing suppuration.

The condition of the blood which contributes to dropsical accumulation is produced by several factors. The loss of albumen and salts has the effect to prevent osmosis into the vessels of fluid in the tissues, which therefore accumulates, and the hepatic derangement and chronic gastric catarrh, which interfere very seriously with digestion and the absorption of its products. The appetite is either wanting or capricious ; food distresses the stomach ; the intestines are filled with gas, the result of the decomposition of certain kinds of food ; and diarrhœa, which nothing controls permanently, comes on toward the close. The continued hyperæmia of the liver causes that appearance known as "nutmeg-liver," the connective tissue undergoes hyperplasia, and the organ, after a period of enlargement, contracts more or less. This state is often confounded with "cirrhosis," but the morbid process is different. The kidneys are affected by the variations in the tension of the vascular system. As a smaller quantity of blood than normal passes through the tufts of the glomeruli, the amount of urinary water decreases, and hence the urine is scanty in quantity, has a high specific gravity, deposits abundantly of urates, and finally becomes albuminous as the tension increases in the venous system. The urine also contains much pigment, but there is rarely any blood present, and there are hyaline casts. The first effect of the persistent venous congestion is enlargement, due to over-production of connective tissue, but in the progress of the case atrophy occurs and the organs become reduced in size, very tough, and dark-purplish in color. These atrophic changes are due to the pressure of the contracting connective tissue and consequent wasting of the proper gland elements. During these alterations the tubular epithelium becomes granular and ultimately fatty, while the basement membrane also undergoes thickening. Infarctions sometimes occur in the kidney during the course of chronic cardiac disease; they are due to obstruction in the branches of the renal artery by emboli; they assume the characteristic wedge-shape, with the apex toward the hilus, and they undergo the same changes as infarctions elsewhere.

Very characteristic cerebral symptoms are also produced by cardiac valvular lesions, but they vary in character according to the valves affected. The disturbed state of the intra-cranial circulation thus occasioned doubtless leads to nutritive alterations in the walls of the cerebral vessels. Furthermore, atheromatous change at the aortic orifice will be followed by similar changes in the intra-cranial arteries. Miliary aneurisms form when the walls of the small arteries undergo these changes. Rupture and consequent extravasation will then take place readily, because of the variations in tension of the blood-vessels. Embolism of the brain is exceedingly common in recent cases of endocarditis. Owing to the position of the left carotid and the left middle cerebral, it is pretty certain that an embolus dislodged from the valves of the left side of the heart will be deposited somewhere within the area of distribution of the left middle cerebral artery. Hence the frequent association of acute rheumatism, valvular disease of the heart, and right hemiplegia, with aphasia. Without causing organic lesions of any kind, very unpleasant and severe symptoms of intra-cranial disturbance are produced by valvular lesions, especially those of the aortic orifice. Narrowing and obstruction, or regurgitation at the aorta, must necessarily produce anæmia of the brain, with the usual symptoms of that condition, as sudden faintness, dizziness, *tinnitus aurium*, persistent headache, etc. Chorea has long been associated with endocarditis. According to the well-known theory of Jackson, chorea is due to multiplex capillary embolisms of the *corpus striatum*, but this view is not generally accepted. In a large proportion—probably in one fourth—chorea is associated with rheumatic endocarditis, but the exact nature of the relation is not now understood.

AFFECTIONS OF THE AORTIC VALVES AND ORIFICE.—The alterations which occur in the aortic valves are very numerous, as respects the character of the resulting deformity. The segments may be adherent by their lateral planes, leaving a central opening through which only the little finger may protrude. A segment may be torn from its base in part or almost wholly.* This accident may result from a suppurating myocarditis, which so weakens the attachment of the valve that it gives way while in the performance of the ordinary functions. Such a degree of shortening and rigidity may ensue that the segments can not successfully approximate, or this change may take place in one or two segments. Besides rigidity and thickening, the valves may be deformed by ragged, dentated, and roughened margins. The margins of the segments may become thinned and slits form, presenting the appearance known as "fenestrated," or the so-called valvular aneurisms may occur, and, giving way, openings are made which render the valve incompetent. Atheromatous changes beginning in the aorta extend downward to the orifices, producing rigidity, narrowing, and deformity. Rough excrescences form and project into the ostium, and so small may it finally become that the smallest finger will barely pass through. The valves also become much altered by calcareous deposits; they become rigid, roughened, and incompetent. As a result of the changes in the valves and orifices—stenosis and insufficiency—the left ventricle is kept too full and the cavity dilates. The septum between the ventricles is pushed over by the distention, encroaching on the right ventricular cavity; the auriculo-ventricular orifice is stretched, and the segments of the mitral are drawn on and lengthened. The increased labor imposed on the muscle of the left ventricle, to propel the blood into the aorta, induces an hypertrophy, and consequently the walls become thicker as the cavity enlarges, although the growth of the walls is not *pari passu*. The papillary muscles are stretched and flattened by the strain of the diastole, and are not hypertrophied.

Symptoms of Stenosis, Rational and Physical.—The character of the pulse has high significance. The ostium being small and the ventricle hypertrophied, the pulse is small, slow, and hard. The sphygmographic tracing exhibits these characters clearly. The ascensional line is rather oblique, the summit rounded, the abscissa low, the descending line oblique, and the interval long; almost the opposite of the tracing in insufficiency. The supply of blood to the brain is insufficient, and hence

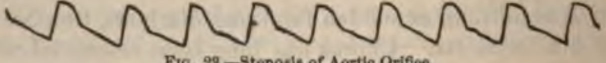

FIG. 28.—Stenosis of Aortic Orifice.

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* Dr. Burney-Yeo, "Lancet," December 5, 1874, "Clinical Lectures on Rupture of the Aortic Valves."

there are attacks of headache, vertigo, syncope, and the patient may fall suddenly relaxed, with or without losing consciousness, or there may occur distinctly epileptiform seizures. The diminution in the quantity of blood passing to the brain may be the cause of serious nutritive derangements in the organ. The left ventricle undergoes dilatation and hypertrophy, and, the mitral becoming incompetent, stasis takes place on the venous side. The lungs are kept abnormally full, hæmoptysis and infarctions may occur, dyspnœa is paroxysmal, and there may be attacks similar to angina pectoris. In the progress of the case the heart becomes less capable of overcoming the resistance, and then, instead of a hard pulse, it becomes soft and weak. On palpation, the apical impulse has the position usual in hypertrophy, but it is much weaker than when there is insufficiency of the valves, and may, indeed, be scarcely perceptible. On percussion, the area of dullness is somewhat increased in the long axis, but little transversely, if at all. Auscultation furnishes a rasping, whistling, singing, or musical murmur, according to the character of the obstruction, and it is systolic in time, audible with greatest intensity in the aortic area—at the junction of the right third costal cartilage with the sternum. It may be very loud and audible a short distance from the patient. If there be regurgitation also, a diastolic murmur will be produced. The diastolic normal sound will be weak because of the diminished elasticity and imperfect closure of the valve-segments. So long as compensation continues there may be no pronounced symptoms, and the heart may be equal to the ordinary duties required of it. When the compensation is ruptured by overwork of the heart, or by the occurrence of disease, then stasis will ensue in the venous system and dropsy will occur. In other cases the amount of obstacle is too great, and the compensation is imperfect; then the disturbances due to the nature of the lesion will slowly develop.

Symptoms of Insufficiency, Rational and Physical.—The pulse has a very different character from that in stenosis. The amplitude of the wave is great, the rise in the beat sudden, its declension rapid. It is known as the "water-hammer" pulse, or as the "Corrigan pulse," from Sir Dominic Corrigan, who described it. The sphygmographic tracing clearly indicates these qualities: the ascent is vertical, the ab-

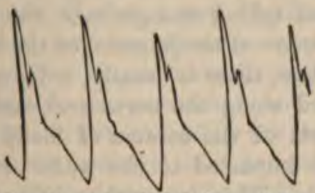

FIG. 24.—Pulse of Aortic Regurgitation.

sciss lofty, the descent abrupt, and, if the case is purely one of regurgitation without other defect, the descent is not marked by the secondary wave produced by the closure of the valve and the recoil of the current. If there is no stenosis, so strongly is the blood propelled into the arteries that small vessels not before visible pulsate distinctly. This condition of things produces the pulsation of the retinal vessels which may be recognized by the use of the ophthalmoscope.

So long as this valvular defect is compensated by dilatation of the left ventricle, and hypertrophy of the walls—excentric hypertrophy—the objective and subjective symptoms are not very pronounced. There are usually a good deal of headache—the pain pulsating synchronously with the heart-beat—more or less dizziness, and pulsation, and *tinnitus aurium*. When associated with atheromatous changes of the intra-cranial vessels, there is great danger of cerebral hæmorrhage. When similar changes have occurred in the aorta and coronary artery, attacks of angina pectoris may take place. So long as the compensation continues unruptured, there will be no difficulty in breathing, no stasis in the venous system, no dropsy ; but, if from any cause the compensation becomes unequal, then there will ensue the ordinary series of phenomena—dyspnœa, cough, enlargement of the liver, congestion of the kidneys, albuminuria, ascites and dropsy. As these cases may continue for years with the lesions compensated, the prognosis is more favorable than in any other form of organic cardiac disease. As soon as the mitral becomes incompetent, dyspnœa begins, the initial symptom, usually, of the widespread disturbance which comes on in the fully developed cases.

In aortic insufficiency, there are present the signs of hypertrophy : the area of dullness, especially the absolute dullness, is increased both in the vertical and transverse diameter, as has been already pointed out in the discussion of hypertrophy of the heart. The murmur proper to aortic insufficiency is a churning, rushing, diastolic murmur, heard at the time and taking the place of the normal murmur, and audible at the aortic area—at the junction of the left third-rib cartilage with the sternum. Also, there is usually, independently of stenosis, a systolic murmur heard along the aorta and carotids, produced probably by the movements of the column of blood in the *dilated aorta*, and by the vibration imparted to the walls of these vessels by the force of the impulsion. This is a rather soft and blowing murmur, not unlike the murmur of anæmia heard in the same situation. It has been shown, further, that a reduplicated sound—systolic and diastolic—is audible in the femoral artery without pressure when there is a marked degree of valvular insufficiency, and it may be developed when there is but little insufficiency, by pressure above and below the stethoscope. This reduplicated sound should not be confounded with the *bruit* which can be produced by pressure of the stethoscope on any artery, and which is a single sound.

Affections of the Mitral Valve and Orifice.—More frequently than at the aortic orifice, the changes in the valves are results of endocarditis—plastic or verrucose endocarditis. Atheroma and calcareous deposition are not such important factors as in lesions of the aortic orifice. Various changes occur in the segments of the mitral. One may become adherent to the ventricular wall; the two segments may be united, the chordæ tendinæ of one segment breaking off; there may be thickening and contraction of each; the borders of the segments may be ragged, thickened by new tissue, and at the same time contracted so as to be quite too small to close the orifice; there may be perforations of the valves by giving way of the so-called aneurisms or by ulcerations, and lastly the valves may unite, leaving a small central orifice. The margins of the ostium may also be thickened and narrowed by inflammatory changes; there may be calcareous deposits, roughening and obstructing it, or the ostium may be enlarged by dilatation of the cavity so that the valves, although normal, are unable to close it perfectly. Insufficiency of the mitral may occur alone, but usually stenosis and insufficiency occur together, and stenosis never, probably, without insufficiency. Whether insufficiency or stenosis, the result is, that the left ventricle is inadequately supplied with blood to distribute through the systemic vessels. The left auricle is over-distended, and the tension in the pulmonary veins is high. The walls of the auricle are hypertrophied, and the endocardium is cloudy in consequence of nutritive changes. The intima of the pulmonary veins is altered by proliferation of its connective-tissue corpuscles, and by fatty degeneration. The pulmonary veins, the pulmonary artery, the right cavities, and the venæ cavæ, are kept over-distended and in abnormally high tension, because the blood is pumped back through, or can not pass through, the mitral orifice, and there is, therefore, ischæmia and low tension in the aortic system.

Symptoms of Stenosis, Rational and Physical.—Having unusual work to do to overcome the obstruction in front, the left auricle becomes hypertrophied. The left ventricle, having less volume of blood to discharge, diminishes in size somewhat, and the aorta also is reduced in caliber, but this is not invariably the case, for there is often either a normal size of the ventricle or it actually becomes enlarged. For example, in a case of mitral stenosis narrated by Balfour, where the segments were "glued together by their margins," and "the opening was so extremely contracted as only to permit the point of the little finger," it is stated that the "left ventricle is slightly hypertrophied, not dilated."* The chief reason why, under a diminished volume of blood, the left ventricle may undergo hypertrophy, is that the contractile energy expended is necessarily increased, because of the obstacles in the circuit. The pulse is small, its tension low, and its rhythm irregular,* but the irregularity is not constantly present, and is a sign rather of rupture of the compensation. There are much cough, difficulty of breathing, bronchorrhœa, often bloody sputa, sometimes hæmorrhage, red-brown induration and hæmorrhagic infarctions; dilatation of the right cavities; general venous stasis, cyanosis; enlargement of the liver, ascites; albuminous urine, and general dropsy. By enlargement and hypertrophy of the left auricle, by dilatation and hypertrophy of the right ventricle, and by the distention of the veins, the stenosis is for a brief period compensated. But the conditions present bring about a slow rupture of the compensation, without the introduction of new disturbances. The changes in the muscular tissue of the right heart, the degeneration of the walls of the dilated vessels, and the alterations produced by the congestion of the liver, intestinal canal and kidneys, suffice to bring on the group of disorders above mentioned, which belong to the mitral lesions. The rupture of the compensation is much facilitated by overwork of the heart, by pulmonary diseases, or by intercurrent febrile maladies. On inspection, rather wide diffusion of the apical impulse is perceived, if there be an apical impulse strong enough for recognition. It is rather a widespread undulation than an impulse at a special point. It extends from within the mammillary line to the right border of the sternum and downward to the epigastrium. It may be absent. On palpation the apical impulse is found to be weak and unresisting, and a purring tremor is felt which may be diastolic or presystolic. If there be regurgitation, a purring tremor may also be felt synchronous with the systole. Both absolute and relative dullness are increased.

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* "Diseases of the Heart," p. 136.

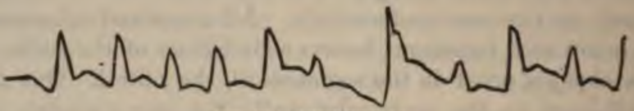

FIG. 25.—Mitral Stenosis.

The transverse dullness is more increased than the vertical, and extends to the right border of the sternum, even beyond, and over the xiphoid appendix. A murmur is audible in the mitral area, of a rather harsh, grating, or blowing character, and occurring with the diastole and extending on up to the systole. The murmur may be presystolic —that is, occurring just before and extending in to the systole, but there are differences of opinion in respect to the time of this murmur. The murmur is usually heard with greater distinctness when the patient sits upright leaning forward, or to the left. No murmur may be audible in some cases under any circumstances. Then the rational signs of mitral lesions possess a high degree of significance, and deserve attentive study, and a failure to appreciate their value and overweening attention to the physical signs are fruitful sources of error, under these circumstances. While, when present, the murmurs are heard in the mitral area with the greatest distinctness, they are propagated toward the apex, and lost toward the base. In a few cases of stenosis, another sign is to be heard over the apex, and at the pulmonary area, namely, reduplication of the second sound. Various explanations of this phenomenon have been offered, but the most probable is that the aortic and pulmonary valves do not close in the same instant of time, owing to the difference in tension of the aorta and pulmonary artery, the tension of the latter being relatively greater and therefore closing before the former. There is a sharp accentuation of the second sound in the pulmonary area, when the reduplication does not occur, owing to the high tension under which the valves are filled and closed. This characteristic of the second sound will disappear when the tension of the vessels declines from any cause or when the tricuspid becomes incompetent.

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* Balfour, "Diseases of the Heart," "Extreme Irregularity," p. 126.

Symptoms of Regurgitation or Insufficiency, Rational and Physical.—So long as the compensation continues, the patient may be comparatively free from discomfort, but the existence of these circulatory derangements leads to pathological changes which effect a rupture of the compensation—e. g., the pulmonary disorders, which are thus brought about, the myocarditis which attacks the walls of the right ventricle, or an intercurrent disease of some kind. Præcordial uneasiness, palpitation, cough, and dyspnœa are the first symptoms experienced when the compensation is ruptured. The pulse becomes soft, small, rapid, and irregular, and while the sphygmographic trace exhibits these features there is nothing distinctive in its form. The legs, presently, become œdematous, the cavity of the abdomen fills, the liver is disordered, the urine is loaded with albumen, and the patient ultimately dies drowned in his own fluids. The physical signs are characteristic. As in insufficiency of the mitral, there is more or less, usually considerable hypertrophy of the left ventricle, enlargement of the cavity and thickening of the walls of the left auricle, hypertrophy and dilatation of the right ventricle; the total result is that the heart is much enlarged, and lies lower and deeper than is the normal condition. The area of dullness, absolute and relative, vertical

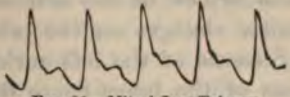
FIG. 26.—Mitral Insufficiency.

and transverse, is enlarged, and the cardiac impulse diffused. On auscultation a systolic blowing murmur is audible in the mitral area, is propagated toward the apex, and may be most intense at the very extremity of the apex. This systolic *bruit* may also, when loud and strong, be heard over the whole cardiac area, and posteriorly under the angle of the scapula ; it may take the place of the first sound, or be heard with it. Usually the murmur can be separated from the proper systolic sound, by very carefully raising the head from the stethoscope so that the ear but touches it. Sometimes the *bruit* is heard with the greatest intensity in the second intercostal space, external to the left border of the sternum, in the position of the appendix of the left auricle, and because of the regurgitating blood like "the fluid in veins producing sonorous vibrations louder at the point of impingement than at that of origin" (Balfour). This, the explanation of Naunyn, is now generally admitted. If there be obstruction as well as regurgitation at the mitral orifice, there will be, as already set forth, a presystolic murmur, extending up to the systole, or under some circumstances a diastolic murmur. In regurgitation, as in stenosis, there is marked accentuation of the pulmonary second sound, until, at least, dilatation of the cavity and incompetence of the tricuspid introduce new conditions.

The diagnosis of mitral disease must rest on a careful survey of the rational and physical signs. Too strict attention to the physical and neglect of the rational signs are frequent sources of error. Exact localization of the murmurs to the areas to which they belong is most important. The history of the case necessarily enters into the question of its nature. When the indications afforded by the history of the case and the rational and physical signs coincide, any serious error is hardly possible.

AFFECTIONS OF THE TRICUSPID VALVE AND ORIFICE.—Only once or twice, in one hundred cases of endocarditis, will the right auriculo-ventricular orifice be the seat of mischief, and then in association with similar changes on the other side of the heart, at the mitral orifice. Stenosis of the left auriculo-ventricular orifice and obstructive diseases of the lungs cause distention of the right ventricle and produce that kind of insufficiency which is known as relative insufficiency. Regurgitation takes place through this orifice, because, being enlarged, the valves become unable to close it during the systole. Over-distention of the auricle and hypertrophy result from the regurgitation, and the tension rises in the venæ cavæ and venous system, while there are ischæmia and diminished tension in the aortic system. The right ventricle also undergoes hypertrophy, because it is filled under the increased pressure of the high tension in the veins and the hypertrophy of the auricle. Regurgitation is often due to changes in structure that are congenital, and stenosis almost always. Very rarely is stenosis produced by acute endocarditis, and, when it does occur from this cause, the anatomical changes are precisely those which have been described as taking place on the other side. The results of stenosis are the same as those of regurgitation, and need not, therefore, be repeated; but stenosis never exists alone, and is always associated with changes on the left side. The pulse is small, weak, but not otherwise altered. A very characteristic symptom is the occurrence of a pulsation in the jugular, synchronous with the cardiac movement. It ought not to be forgotten that waves are caused in the jugular by the respiratory movement—by the expiratory pressure. The true venous pulse does not extend beyond the bulb of the jugular, if the valves of the vein are intact, but by distention they become so, when the venous pulse is perceived along the whole extent of the vessel, extending even to the external jugular. It is synchronous with the contractions of the heart. The pulsation may be double, produced by the contraction of the auricle, and by the beating of the aorta, the vena cava superior lying in close proximity to that vessel. There is a feeble venous pulsation when there is regurgitation at the mitral orifice, a stronger one with coincident insufficiency of the tricuspid, and with the latter alone. That this pulsation is produced by the lesions above mentioned, and is not an oscillation in the blood-current caused in the various ways already described, is determined by merely compressing the vessel with the finger, when the following facts will be elicited: If the pulsation be due to the heart-movements (regurgitation), when the vein is compressed at its middle, it will continue below the point of compression and cease above; if due to the beating of the carotid, it will continue above the point of compression, and cease below. If due to the respiratory movements, the pulsation will be synchronous with those movements; if to the heart-movements, sychronous with them; if respiratory, they will cease with the suspension of breathing; and, if cardiac, will continue. There is an equally characteristic venous pulse of the liver, which is felt immediately on the occurrence of the changes on the right side of the heart, because the hepatic veins are not provided with valves. The pulsation, synchronous with the cardiac movements, may be felt over the whole organ, or be confined to the right lobe. The venous pulsation in the neck may appear and disappear under the variations in the fullness of the right cavities and the force of the ventricular contractions. The hepatic pulsation is affected by effusions in the abdomen, as well as by the state of distention of the vena cava and the hypertrophy of the right ventricle. So long as the valves of the jugular remain intact, the increased tension under which their closure is effected causes a murmur, humming and clacking combined, which is audible in the bulb. The hypertrophy existing chiefly to the right, the area of impulse must be seen to the right, and is rather diffused.

Dullness on percussion, due to the enlarged right auricle, can be developed to the right of the sternum from the second to the fourth rib, and the dullness due to the right ventricle, to the base of the sternum, to the xiphoid appendix, and to the central and right portion of the epigastric region. A pulsation produced by the right auricle can be seen and felt sometimes in the right, second intercostal space. On auscultation in the tricuspid area—the lower segment of the sternum—we hear a blowing murmur, systolic in time, and most intense at the junction of the intercostal space between the fourth and fifth rib and the sternum; sometimes, most intense over the xiphoid appendix. This is the characteristic murmur, but there are associated with it the valvular mitral murmurs which almost always are present, and are audible with the greatest intensity at the mitral area and toward the apex. These are both systolic, presystolic, and diastolic, as has been pointed out. In the affections of the right auriculo-ventricular orifice, the pulmonary second sound is weak, because of the diminished tension in the pulmonary artery, unless there is coincident obstruction or regurgitation at the mitral orifice, which causes an accentuation of the pulmonary second sound. The mechanical effect of the lesions on the right side is immediate, and compensation is possible to a very limited extent. Extreme venous stasis soon occurs, with the attendant symptoms of hepatic disturbance, ascites, albuminuria, general dropsy. The prognosis is therefore unfavorable. The diagnosis is difficult because of the coexistent mitral lesions, but the lesions of the right auriculo-ventricular orifice are established by the determination of these physical signs: a well-marked, true venous pulsation of the neck; a systolic murmur, audible with the greatest intensity at the junction of the intercostal space between the fourth and fifth rib with the right border of the sternum, and a weak, pulmonary second sound.

AFFECTIONS OF THE PULMONARY VALVES AND ORIFICE.—These may be congenital or acquired. When acquired they are produced by endocarditis, or are due to calcareous deposition and atheromatous degeneration, but acquired changes are extremely rare. The results of stenosis and insufficiency are the same, and consist of dilatation of the cavity and hypertrophy, leading to insufficiency of the tricuspid. In *insufficiency* of the pulmonary valves the resulting conditions are the same as in the corresponding change at the aortic orifice. The pulmonary artery and its divisions undergo dilatation, the intima becomes the seat of the nutritive changes already described, and lobular pneumonia and hæmorrhagic infarctions occur in the lungs. The rational signs are dyspnœa, deficient aëration of the blood and cyanosis, distention of the superficial vessels, dropsy, palpitation of the heart, præcordial oppression, sudden attacks of suffocative feeling, with præcordial pain and intense anxiety, etc. The physical signs are those of enlargement of the right cavities, a loud diastolic murmur heard with great intensity at the left border of the sternum and the upper margin of the third rib, and propagated toward the middle of the sternum, opposite the fourth rib and downward, and is lost going toward and over the great vessels at the base. There may be also a systolic murmur. These symptoms only occur when the compensation is ruptured, for the hypertrophy of the ventricle walls and the dilatation of the cavity compensate very fully for the mischief done.

Stenosis is a more important condition than insufficiency, but it is congenital stenosis with which we have to deal chiefly, the acquired condition being exceedingly rare. In congenital stenosis the changes consist in constriction of the pulmonary artery, unclosed foramen ovale, unclosed ductus Botalli, stricture at the ductus Botalli, with hypertrophy of the right cavities. The importance of these congenital defects, besides the damage to the heart, consists in the frequent association of these anatomical anomalies with tuberculosis of the lungs. The right ventricle enlarges to a remarkable extent, the walls attaining in thickness to the dimensions almost of the left. The result is, there are present the physical signs of hypertrophy of the right ventricle—an increased area of cardiac dullness to the right; a blowing, systolic murmur, audible in the pulmonary area, and propagated not toward the base and great vessels, but somewhat to the left and a little downward, the point of greatest intensity being the junction of the third rib, upper border, with the left border of the sternum; weak or inaudible second sound. The rational symptoms correspond to the anatomical conditions. The compensation effected by dilatation and hypertrophy of the right ventricle suffices to maintain a condition of comparative comfort, but unusual physical exercise, obstructive pulmonary diseases, and other causes bring about a rupture of the compensation, when there ensue difficulty of breathing, cough, cyanosis that may be very intense, but general dropsy and albuminuria occur only when the right ventricular wall weakens by myocarditis.

The duration of these cases of congenital defects in the structure of the heart varies with the degree of deformity and the circumstances in life. The compensation may be so perfect that the heart is equal to the needs of a quiet existence, and comparative comfort may be enjoyed by youths who possess even a considerable degree of cyanosis. But the degree of cyanosis is usually a measure of the success of the efforts at compensation. The subjects of congenital pulmonary stenosis are otherwise imperfect in organization—they are comparatively weak, develop slowly, have soft, flabby muscles, bones do not unite, and the nutrition continues poor. Beside the cyanosis, which is usually most strongly marked in the extremities, they have cold hands and feet, and possess but little endurance of cold, are subject to asthmatic attacks, to giddiness and vertigo, to epileptoid attacks, etc. The duration of life in these congenital cases varies from a few months to twenty or thirty years.

Treatment.—We quite agree in the statement of the late Dr. Flint,* that caution is necessary in the expression of opinion to the subjects of cardiac mischief; the importance of recognizing the fact that some murmurs have no pathological nor clinical significance; the good results obtained from the treatment of associated morbid states in cases of undoubted valvular disease; and, finally, the striking relief derived from the timely use of "digitalis and active hydragogue purgation repeated from time to time."

When the mischief done to the heart is recent, and the newly formed connective tissue is contracting, it is highly important to give the heart "physiological rest," to enable the damage to the valves to be repaired as completely as can be effected. The rest is best secured by maintaining the recumbent posture much of the time during the period of convalescence, by the careful administration of veratrum viride, or chloride of barium, to keep the revolutions of the heart at about fifty to sixty per minute, and by iron and suitable diet to improve the quality of the blood, or by the use of nitro-glycerin to lessen the work of the heart in overcoming the peripheral resistance, and to improve the nutrition of the organ when the general vascular tension is low. When compensation is effected and the heart is equal to the task of overcoming the obstacles, no medicinal treatment is necessary. Every effort must be directed to the maintenance of the compensation, by quietude of mind and body, and by avoidance of all causes of diseases. Nevertheless, daily open-air exercise and exposure to sunshine are necessary to maintain health at the proper standard; for, if the blood is impoverished by an in-door life and the want of appetite and imperfect sleep, which are necessary results, the rupture of the compensation must then take place. Formerly severe exercises were strongly condemned as hurtful to a weak heart, but now, by some medical extremists, are as strongly approved as means to promote the nutrition and to increase the muscular power of the organ. In the natural course of events in valvular affections, the nutritive alterations which occur in the tunics of the vessels and in the heart-muscles ultimately effect a rupture of the compensation. Anæmia not only hastens the pathological processes taking place in the vessels and in the heart, but it actually inaugurates similar changes. It is, therefore, a measure of the highest importance to keep the appetite, digestion, and blood-making process in the most efficient state. When the heart is behaving badly in consequence of the anæmic condition, the organ is relieved by attention to the nutrition. Unless, therefore, under such circumstances there is plain need of digitalis and the other "heart-tonics," so called, they should be avoided, for they disturb the stomach and interfere with digestion. When, in women especially, the compensation is not ruptured, but great distress is experienced from anæmia or the chlorotic state, the indications clearly are not to treat the heart, but those nutritive disturbances on which the functional troubles depend. When such subjects are not relieved by stomachic tonics, iron, and a generous diet, the system of rest, forced feeding, massage, and muscular faradization proposed by Weir Mitchell may be resorted to with advantage. Besides the measures necessary to prevent or overcome anæmia, the dietetic management requires the patient with compensated valve-mischief to avoid such cardiac stimulants as tea, coffee, tobacco, and alcohol in any form, except a little wine allowed at dinner provided it improves digestion. The choice of a suitable chalybeate can be made from a long list of preparations. It is a rule that combinations of iron with a mineral acid are more effective and often better borne than the milder and supposed more easily assimilated citrates, tartrates, and carbonates. German therapeutists much prescribe the ethereal acetated tincture of their pharmacopœia. The tinctura ferri chloridi is, probably, the most generally useful and efficient of the ordinary preparations. It should be given always well diluted with water after meals, and should be taken through a glass tube or a straw. An excellent stomachic tonic is tincture of nux vomica—ten drops to twenty—*ter in die* and before meals, or the milder tinctures of colomba or gentian may be preferred. A combination of great value in these cases is the elixir of the phosphates of iron, quinine, and strychnine (Aitken). The nutrition in cases of compensated valvular lesions often fails slowly, from the gradual congestion of the liver and of the intestinal mucous membrane. The digestion is slow and insufficient, the appetite fails, and the absorption of aliment is seriously interfered with by the hyperæmia and distention of the vessels. Timely recognition of this state and the use of appropriate means will prevent serious trouble. Excellent remedies are arsenic, euonymin, and salol, and under some circumstances, calomel; they are stomachic tonics, and, in sufficient quantity, powerfully stimulate the hepatic functions and deplete the portal system. The treatment should be commenced by free action of the intestines procured by the compound jalap powder, elaterium, podophyllin resin, and similar agents. Then stomachic tonics, chalybeates, and digestives, as pepsin and lactopeptine, are indicated. The kidneys should be kept active, and this is best accomplished by the simultaneous but not conjoint use of a chalybeate and a diuretic, as tincture of iron and solution of bitartrate of potassa—the iron to be taken after meals, and the potassa solution to be drunk freely between meals. An excellent method of managing these cases, when a rupture of the compensation is threatened, is to give two or three times a week some efficient doses of iridin or euonymin, and to prescribe iron, quinine, and digitalis in pill-form—a half grain of ferrum redactum, three grains of quinine, and a grain of digitalis in a pill three times a day. If the stomach is doing fairly good work, the best results may be expected from this combination. The practitioner is usually consulted when the failure of the heart, dyspnæa, cough, anæmia, albuminuria, and beginning dropsy, announce the rupture of the compensation. The principles of treatment differ somewhat, according to the seat and character of the lesion and the condition of the system. As the ultimate effect of all cases of valvular disease of the heart is to cause ischæmia of the arterial system and stasis of the venous, a general method of therapy may be first developed and the special indications pointed out subsequently. The remedy which has been most employed to oppose the condition of the vascular system in mitral disease of the heart is digitalis. In prescribing this agent there are several points to be carefully considered. Is the digitalis of two years' growth? Is it English or German? Is it wild or cultivated? The second-year plant contains more of the active principle; the production of this continent seems inferior to that of English or German sources; the wild digitalis is more active than the domesticated. For the effect on the circulation and on the kidneys, the officinal infusion is to be preferred to the other preparations, but the infusion is only serviceable when it is made from the proper digitalis. It must be given in sufficient quantity to produce its physiological effects—to diminish the number but increase the force of the pulsations; to raise the tension of the vessels; to increase the urinary discharge. The higher the tension at the periphery, the more decided the recoil, and consequently the better filled is the coronary artery, which includes a more active and healthy state of nutrition of the cardiac muscle. The higher tension of the vessel means an arrest of the outflow of the serum and more active absorption. When the compensation is ruptured, the digestive organs suffer and the blood-making is inefficient. Excretion by the liver is hindered, and the waste of albumen through the kidneys lessens rapidly the amount of this important constituent in the blood. The poverty of the blood reacts, again, on the circulation through the heart. When, therefore, the necessity for digitalis arises, the demand for iron and bitter tonics (quinia) must be heeded also. Experience has abundantly demonstrated that the effects of digitalis are more decided and more lasting when iron and quinia are given at the same time. A tablespoonful of the officinal infusion three times a day until the characteristic effects are produced, and then twice a day, is the amount usually required and that can be borne. As its action is slow, frequent repetition of the dose may cause serious symptoms. If large doses are taken, and if the pulse is much reduced, the patient should maintain a fixed position—what position soever it may be—and not change it suddenly. Especially should he not rise suddenly from the recumbent posture, for under these circumstances the pulse becomes rapid and feeble and the surface cyanosed. When headache, dizziness, disturbances of vision, vibration of external objects, and anxiety are produced, the dose must be at once reduced or discontinued. It should also not be forgotten that digitalis continued in large doses affects the motor power of the heart ultimately, by exhausting the irritability of the ganglia, when the action becomes rapid, weak, and irregular. It is good practice, during the long-continued use of digitalis, to suspend it for a few days at a time. If it can not be borne, cimicifuga may be substituted—a half to a drachm of the fluid extract three times a day. Sufficient attention has not been given to the utility of cimicifuga as a cardiac tonic and substitute for digitalis. Caffeine and convallaria are more active agents, which more nearly approach digitalis in power, and may be substituted for the latter when its effects on the stomach require it to be discontinued. In antagonism to some of the actions of digitalis, and as a means of lessening the work of the heart, we can not speak too highly of nitro-glycerin. Of the mineral tonics, no one is so serviceable as the acetate of lead. When there is much oppression of breathing, the patient unable to lie down, and becoming exhausted from loss of sleep, no remedy is so valuable as morphine hypodermatically. It affords surprising relief to the distressing symptoms, improves remarkably the driving power of the heart, causes free diaphoresis, and gives time for the action of the other remedies. From the $\frac{1}{12}$ to $\frac{1}{4}$ of a grain of morphine, according to the character and susceptibility of the patient, should be given. Next to the remedies for the heart, in importance, are the hydragogue cathartics. The greatest relief is afforded by draining off fluid from the intestinal mucous membrane. Euonymin and iridin have already been mentioned, but more powerful remedies are necessary when there is general dropsy. One of the most useful and efficient of these is the compound jalap powder. As it is important not to interfere with the digestion, this remedy should be administered in the early morning. If not sufficiently active, podophyllin may be added, or, this failing, elaterium may be substituted. Free transpiration by the skin should be maintained. This is best effected by the vapor-bath. The mistake must not be made of attempting to act on the skin and kidneys at the same time. When digitalis is being taken, and bitartrate of potassa or other diuretics, the skin must not be excited at the same time ; on the other hand, free purgation assists the action of diuretics. When digitalis can not be borne by the stomach, it may act quite efficiently by external application to the abdomen or back : some leaves inclosed in a muslin bag are steeped in warm water, and kept applied for several hours. When the vapor-bath can not be used, a good substitute is a warm wet-pack covered with blankets. Remarkable benefit has been obtained from the treatment by compressed air, and by the inhalation of oxygen. The compressed-air treatment diminishes the tension in the venous, and elevates it in the aortic system, and also gives relief by contributing to the oxygenation of the blood. Oxygen merely acts in the latter mode, and often affords great comfort when there are paroxysmal attacks of dyspnœa. There are some limitations to the use of digitalis in ruptured compensation with its direful results. It can not be borne at all by some subjects. It is contraindicated in aortic stenosis, and may be dangerous in large doses. When there is mitral insufficiency, as well as aortic stenosis, digitalis may be given, but only in small doses, with a view to its diuretic action. Again, digitalis is of doubtful utility if not positively contraindicated in fatty heart, and consequently in cases of dropsy from dilatation and insufficiency due to fatty degeneration.

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* The "Medical News and Abstract," January 7, 1880.

HEART-CLOTS.

Definition.—By the term *heart-clot* is meant a mass of fibrin or of coagulated blood found in one or more of the cavities of the heart. They are divisible into three varieties: First, translucent masses of fibrin, soft, yellowish, and full of serum, loosely attached to the chordæ tendinæ, trabeculæ, or other projecting parts; second, large, loose, black coagula occupying the right ventricle or auricle, and extending into the pulmonary artery or venæ cavæ; third, coagula of variable size, attached to projecting parts, found in all cavities, but chiefly in the left ventricle, and consisting of coagula containing a puriform-looking fluid in their interior. The first variety is not pathological, is formed during the death-agony or after death, and is found in the subjects of chronic wasting disease. The second variety may or may not be pathological, and stand in a genetic relation to the suspension of the cardiac movements. The third variety is always pathological.

Causes.—The occurrence of these clots is not affected by sex, but they are more frequent at the middle period than at the extremes of life (Bristowe*). There are two leading factors in their causation—a condition of the blood; disease of the heart itself. In many diseases the fibrinogenous substance seems to be greatly increased, and thus a state of ready coagulability is induced. If, under these circumstances, the coagulation of the blood is favored by a slow and feeble action of the heart, a slight cause suffices to determine it. The actual determining cause is disease of the heart itself, roughness of some projecting part, or fibrinous concretion deposited on such rough surface.†

Pathological Anatomy.—Clots are found in all the cavities of the heart, but most frequently in the left ventricle and least frequently in the left auricle (Bristowe). They form in by-places, and are entangled in the rough surfaces and inequalities. The appearance of the clots differs according to the circumstances of their formation. Leaving out of consideration the masses of fibrin, which have no pathological import, the two other varieties differ in consequence of the changes wrought by age. The second variety mentioned above consists of a large, black, rather loose venous coagulum, which fills one or the other cavity of the right side and projects into the annexed vessel, which may be completely filled by it. Such a clot, we may suppose, is sometimes the cause of death after *post-partum* hæmorrhage, or such as Sir Joseph Fayrer describes* as forming and causing sudden death after surgical operations. After profuse hæmorrhage of this kind, the propelling power of the right ventricle is so feeble that coagulation may readily ensue. The shock of a surgical operation may induce such slowness and weakness as a severe hæmorrhage, and result in the same accident. In the third variety the clot has undergone transformations due to age. It is firm, tough, grayish, yellowish, and brownish in strata, or variously intermingled, and attached to the columnæ carneæ, chordæ tendinæ, or other parts. It usually contains in the interior, in a pseudo-cyst, a quantity of thick fluid having a "grumous" or "puriform" appearance, and consisting of the fibrin, red and white corpuscles, undergoing the transformation usual to blood under these circumstances.† These clots are in position for a long time, often. Rarely are they found in a sound heart, and usually the changes of endocarditis have taken place, the coagulation of the blood being induced by roughening and exudation of the membrane.

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* "Pathological Society's Transactions," vol. xiv, p. 71.

† Ibid., cases by Dr. J. W. Ogle.

Symptoms.—Nothing can be more indefinite than the symptomatology of heart-clots. Nevertheless, we may make an attempt to define, from recorded cases and from observation, the character of the disturbances of function caused by them. There are two distinct groups of symptoms belonging to the two forms of clot. After *post-partum* hæmorrhage, or after a surgical operation, or during the course of some septic disease, there suddenly comes on an extreme oppression of breathing, wild restlessness, beating about the bed and crying out for air, deep cyanosis, a fluttering heart without pulse at the wrists, which stops in a few minutes; the patient falls back, the agitation ceases, but then a general convulsion may occur, and all is over, or death occurs quietly without any convulsive movement. In the other variety the symptoms develop more slowly, and may extend over several weeks. The earliest symptoms are irregularity in the heart-movements, indistinctness of the murmurs, difficulty of breathing, anxiety, oppression, cyanosis. The action of the heart becomes more and more feeble, the sounds run into each other and are dull and confused, the difficulty of breathing continues, moist *râles* appear all over the chest from œdema of the lungs ; the cyanosis deepens ; dropsy comes on ; stupor passing into unconsciousness, and convulsions end the scene.

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* "The Medical Times and Gazette," vol. i, 1873, p. 58; also "Pathological Society's Transactions," vol. xxvii, p. 70.

† Cases by Dr. J. W. Ogle, "Pathological Society's Transactions," vol. xiv, p. 65, *et seq.*

In most of the cases recorded by Ogle, the urine was albuminous ; there were lesions of the lungs, and effusion into the thoracic cavity. While the recorded symptoms are closely similar to the account given above, the state of the heart as to rhythm and the character of the sounds differ among themselves, and agree in part only with the above description. The duration of these cases ranged from a few days to six weeks, and the symptoms during that time seemed to depend on the presence of the clot found *post mortem.*

Treatment.—Notwithstanding the uncertainty which must attend the diagnosis in these cases, which at its best must be a fortunate guess, some details of treatment are necessary. The treatment by frequent small doses of ammonium carbonate offers the best prospect of relief. In the cases which occur suddenly, and immediately extinguish life, the intra-venous injection of ammonia should be practiced. This method consists in the injection into any vein—in this case, the jugular—of one part of aqua ammoniæ to two parts of water, by an hypodermic syringe. Of course, precautions must be taken to avoid the introduction of air or any foreign body. It has been abundantly demonstrated that this intra-venous injection of ammonia is entirely safe. In the less acute cases, there is a small prospect of success from the persistent use of the ammonia. The action of the heart must be maintained by the judicious use of digitalis and alcoholic stimulants.

PALPITATION OF THE HEART.

Definition.—By the term *palpitation of the heart* is meant a functional disturbance of the organ, characterized by increased rapidity of movement, with more or less irregularity of rhythm.

Causes.—The heart possesses a power of independent motion ; but as this motor apparatus is not sufficient to keep up the action of the organ, it receives accessions of force from the great centers. To maintain the movement at a uniform rate, there is a regulator apparatus, designed to prevent overaction, or "to inhibit." Besides this mechanism for evolving force, and applying it so as to produce uniform results, the action is affected by the state of the vessels, by the density of the blood, by the movements of the respiratory organs, by the activity of the organic functions in general, and by the functions of animal life. Accordingly, to maintain the action of the heart, there are—1. A motor apparatus—rhythmically discharging motor ganglia—situated in the substance of the heart. 2. Excitors of activity, branches from the cervical sympathetic, and also from the spinal cord, irritation of which increases the movements of the heart. To regulate the movements of the heart, there are—1. The pneumogastric, irritation of which may arrest the heart in the diastole. 2. The depressor nerve of Ludwig, which acts by dilating the blood-vessels. The fibers of the sympathetic, dilator, and constrictor, affect the work of the heart by increasing or lessening the tension at the periphery. When the peripheral vessels are dilated, the work to be done by the heart lessens, and hence the contractions are less numerous and forcible, and *vice versa*.

The mechanism by which the action of the heart is kept at a uniform rate may be disturbed by a variety of causes: by muscular exercise; breathing rarefied air, as in the ascent of mountains; by mechanical interference with the movements of the organ, as thoracic effusions, tumors of the mediastinum, flatulent distention of the stomach, athèroma of the arterial system generally, etc. Moral and emotional causes, as grief, hope, anxiety, fear, excessive mental effort, etc., increase the action of the heart. Various reflex troubles have the same effect—as affections of the nervous system, reacting on the nervous apparatus of the heart—such as uterine disease, gastralgia, worms in the intestinal canal,* etc. The cardiac ganglia are rendered irritable by the excessive use of tea, coffee, tobacco, spirits, etc. The excitor apparatus of the sympathetic may be the seat of a disturbance, as in Grave's disease, etc.

Symptoms.—There may or may not be, previous to the attacks of palpitation, any symptom of trouble in the heart. When such preliminary symptoms are felt, they consist of a vague sense of uneasiness, præcordial oppression, or dull pain. There is no fixed period for the attacks, unless excited by some habit or custom, as eating, smoking, etc.; neither have they any special duration, but may last from a few minutes to some hours, or a day. The attack consists of a rapid and tumultuous beating of the heart; dyspnœa, anxiety, and an hysterical sense of choking accompany the beating; the heart seems almost to turn over, to rise up into the throat; the recumbent posture can not usually be borne, especially lying on the left side, and the sitting posture, leaning somewhat forward, is the most comfortable position; there are also experienced more or less vertigo, faintness, flashes of light, coldness of the surface with cold sweating and a very weak pulse, or it may be the surface is warm and perspiring, the pulse full and strong. The face may be pale or flushed, but is always expressive of anxiety; speech is difficult, or is arrested. The physical exploration, if no cardiac lesion exist, is merely negative. The movement, if very rapid, can not be separated into its component parts. Examination must be made, in the interval of the seizures, to ascertain the real condition of the heart. The duration of the attacks, as already stated, is very variable. The beating may subside in a few minutes, or several hours may be occupied in returning to the normal. At the conclusion of the paroxysm, a quantity of pale, limpid urine is usually passed, and there is a strong sense of fatigue and exhaustion, with a tendency to sleep.

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* Case of Dr. Cotton ("The British Medical Journal," June, 1867), in which the pulsations were 240 per minute, and ceased on the evacuation of a tape-worm.

Treatment.—Prophylaxis is important. The vice, of whatever kind, on which the attacks depend, should be removed. Tea, coffee, and spirit drinking must be given up ; errors of digestion, reflex disturbances, and curable diseases must be corrected or cured. The hygiene of the individual must be carefully investigated, and sources of disturbance be put aside. The general health must be maintained at the highest point of efficiency. In the absence of any explanation of the paroxysms, the presence of a tape-worm may be suspected. For the immediate relief of the paroxysm, there is no remedy so efficient as the hypodermatic injection of morphia. If the surface is pale and the extreme vessels contracted, inhalation of nitrite of amyl (two or three drops) affords prompt relief. The inhalation of ether is also effective. All narcotic agents must be used with caution, because of the certainty, if the attacks are frequent, that the habit of their abuse will be formed. The application of cold, in the form of an ice-bag to the præcordial space, is an effective means of quieting the heart. The galvanic current, from ten to thirty or forty elements, passed through the pneumogastric and cervical ganglia of the sympathetic, often gives great relief. If there is no cardiac disease, chloral is an efficient quieting agent, and the bromides may also be given with good results.

DISEASES OF THE BLOOD-VESSELS.

ARTERITIS—INFLAMMATION OF THE ARTERIES.

Definition.—The acute form of arteritis is uncommon, and is rather a surgical than a medical topic. Chronic arteritis, on the other hand, is not only a common but it is an extremely important disease. It has received various designations, as *endarteritis*, *atheromatous arteritis*, *arterial sclerosis*, *arteritis deformans*, etc., intended to indicate the nature of the change undergone by the vessels.

Causes.—It is extremely rare before forty, and frequent after fifty. Men are probably more liable to it than women, but there is slight difference as regards sex. Various cachexiæ seem to hasten its development. A fatty change occurs in the intima during the course of severe and prolonged anæmia. Chronic alcoholism, the poison of lead, gout, rheumatism, syphilis, etc., are supposed to be influential in developing the disease at an early period. Functional strain, in accordance with a well-known law, tends to excite arteritis; hence its early appearance in the aorta. Sometimes aortitis is derived, by contiguity of tissue, from endocarditis.

Pathological Anatomy.—The initial change consists in a proliferation of the connective-tissue corpuscles of the intima; the young cells crowd the space between the lamellæ, and, pushing up the intima, form a projection about a line above the general level. This abundant formation of new cells requires an amount of pabulum which can not be supplied, and hence the proliferating cells undergo a fatty degeneration. While this process is going on, a solution of the basis substance (the connective-tissue matrix) takes place.* This change appears to the naked eye as yellowish or yellowish-white opaque spots or patches, distributed through the thickened elevations of the intima, which become soft and friable, and are gradually detached, leaving an abrasion, or "atheromatous ulcer." These abrasions may be coated with masses of fibrin, or blood-clot may form on and adhere to them. Coincidently with the process of fatty metamorphosis, another process, beginning also in the sclerosed intima, develops. This consists in a deposition of calcareous material—the lime salts, chiefly—in the basis substance of the intima, and between the lamellæ. Plates of considerable size are thus formed in the aorta; they may be several inches in length, and of a curved shape corresponding to the aortic curve, and may extend over one half, even more, of the circumference of the vessel. Their rough surfaces project through the innermost lamella into the vascular lumen. These two processes very frequently coincide. The alterations taking place in chronic arteritis are not confined to the intima, but the media and the adventitia also participate. The unstriped muscular fiber undergoes fatty metamorphosis and calcification, or disappears by simple atrophy. In advanced cases the adventitia inflames, becomes infiltrated with cells, or undergoes fibroid degeneration. The results of arteritis are very important; when the small vessels are affected, their lumen is encroached on and may be entirely obstructed, or a large number affected to a less degree, the amount of blood passing to the district supplied by them will be much reduced, and important nutritive alterations must occur. The changes in the tunics of the vessels especially involve their elasticity, and they become mere rigid cords, through which the blood passes in jets. The loss of the power of elastic recoil exposes them to injury as the blood is driven through, and they slowly dilate or yield in places, forming sacculi, or are torn outright. The increased resistance to the propulsion of blood, caused by these changes in the arteries, leads to dilatation and hypertrophy of the left ventricle. Named in the order of relative liability to arteritis deformans, are the aorta, the cerebral arteries, the coronary, the arteries of the extremities, and, lastly, the arteries distributed to the organs of vegetative life.

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* Rindfleisch, *op. cit.*, p. 211, *et seq.*

Symptoms.—The symptoms are obviously of a very diverse character when produced. Nothing is more usual than to see men after fifty with extensive atheroma, without a single symptom referable to it. Nevertheless, numerous and important consequences follow arteritis in some situations, and at certain stages of its development. Arteritis of the aorta, and the cardiac disturbances due to it, and arteritis of the brain, and the structural alterations produced by it, are the same as regards the arterial change, but are widely different in respect to the symptomatology. If the lumen of the aorta is encroached on, especially if very great narrowing takes place at the bifurcation of large arteries, or if extensive arterial districts have undergone sclerosis, the work of the heart to distribute the blood is so much increased that the organ undergoes hypertrophy. This change is indicated by the heaving impulse, by an extension of the area of cardiac dullness downward and to the left, and by accentuation of the second sound. Murmurs, due to regurgitation or stenosis, or both, may be audible with greatest intensity in the aortic area, when an extension of disease from the aorta to the semilunar valves, or to the endocardium, takes place. Weakening of the heart, dyspnœa, general œdema, may finally occur from degenerative changes in the heart-muscle, the result of atheroma and calcification of the coronary artery. The physical signs, then, of hypertrophy, from the causes above mentioned, must necessarily disappear and be supplanted by others when the aortic valves and the cardiac tissues become diseased. Dilatation of the ascending aorta may produce a pulsation in the right second intercostal space that may be mistaken for aneurism, and, if the dilatation be considerable, some dullness on percussion may be developed in the same position. The changes of arteritis deformans may be studied clinically in some superficially placed arteries, as the radial and the temporal; they are rigid, tortuous, irregular in size, and may be rolled under the skin like whip-cord. The tortuosity is increased during the systole, and lessens during the diastole, and the pulse is delayed—firm when the calcification is beginning, but becoming less and less recognizable as the artery degenerates into a calcareous tube. The loss of elasticity of the arterial tunics influences the sphygmographic tracing, which exhibits the same features as in albuminuria—rounded summits, oblique descent, without dicrotic or recoil wave. Advanced endarteritis leads to disastrous results in the nutrition of peripheral parts—the fingers and toes. In consequence of the diminished supply of blood, the sensibility is low, the skin bluish, benumbed, and cold, and the least injury may set up destructive inflammation. A thrombus forming in the principal artery, dry gangrene will follow in the parts below, or in a small vessel of the foot; a single toe, or several toes, may slough off. Even more serious results follow endarteritis of the internal vessels. Thus, as has been pointed out in the article on gastric ulcer, solution of the mucous membrane and the subsequent formation of a chronic ulcer may have its origin in disease of an artery and thrombosis. It is a singular fact that, although the arteries of the vegetative organs are the last to be invaded by endarteritis, yet it occasionally happens that a small part of an artery supplying the gastric mucous membrane is the seat of this degeneration, with the disastrous effect above mentioned. But the arteries of the brain are much more widely and early affected by endarteritis than of any vessels except the aorta, and indeed this morbid process may begin in the brain. The dilatations of the arterioles and small arteries, known as miliary aneurisms, are the great cause of cerebral hæmorrhage; thromboses of the capillaries and small arteries induce local softening; endarteritis, without interrupting the passage of the blood through the lumen of the vessels, impedes the transference of the nutritive materials to the tissue of the brain, with the result of serious impairment of the nutrition of the organ, and consequent failure of mental power, and the usual objective evidences of cerebral mischief.

Course, Duration, and Termination.—The course of endarteritis is influenced by various circumstances. The progress of the change is hastened by the abuse of spirits, and by such cachexiæ as syphilis, rheumatism, and gout. It is very chronic, and its duration may be measured by years. As has been pointed out, many cases exist without causing any disturbance; others are very important in consequence of the lesions invited by arteritis. The termination is a question of the nature of the secondary lesions, and especially of the changes in the cerebral arteries. There is more danger in those cases occurring at an early period of life. For example, the author has seen life terminated by a small aneurism of the basilar artery, when this was the only spot where endarteritis existed.

Treatment.—Although, when the change has once taken place in an artery, nothing can be done to remove it, the author believes that the progress may be, if not arrested, at least retarded by proper treatment. There are three remedies of special importance in this disease; quinine, hypophosphite or lactophosphate of lime, and cod-liver oil. The phosphite or phosphate of lime, and the cod-liver oil, should be given, after meals—a teaspoonful of the sirup of either phosphate or phosphite, but preferably of lactophosphate of lime, and a teaspoonful of cod-liver oil. They may be given in an emulsion simultaneously, or one may follow the other, and they should be taken without failure for months at a time. Quinia should be given in five-grain doses, morning and evening, on alternate days at various times. Personal habits contributing to arterial degeneration should be discontinued. A syphilitic taint should be corrected, and lead or other poison deposited in the tissues should be eliminated. The diet should be composed of nutritious materials, but indigestion ought to be avoided. Daily outdoor air and moderate exercise are very necessary hygienic measures.

ANEURISM OF THE AORTA.

Definition.—An *aneurism* is a tumor formed of the coats of an artery, and containing blood and fibrin. They are designated *cylindrical*, *fusiform*, or *sacciform*, according to their shape; and *true* if all the layers are engaged, *false* if one or two form the walls of the sac. A *dissecting aneurism* is one in which, the intima and media giving way, the blood dissects along underneath the adventitia, and the walls of the sac are composed of this membrane only. A *varicose aneurism* is one in which a communication is established with the venæ cavæ, the innominatæ, the right auricle, or the pulmonary artery. The anatomical distinctions on which these names are based are important chiefly from the prognostic point of view.

Causes.—The aorta is the favorite site of aneurisms, because, in the performance of its functions, it is subjected to great strain. If the left ventricle is hypertrophied, the blood-pressure in the aorta is increased, and the tendency to the formation of aneurism is greater. Powerful muscular effort has the same effect, and hence those who are engaged in occupations requiring the exertion of their utmost strength suffer more from this malady than those having easier pursuits. Men are more liable to the disease than women, and for the same reason that those who labor hard suffer more. The frequent association of syphilitic infection and aneurism has attracted much attention, but a causal relation has not yet been established. Chronic arteritis is, doubtless, the chief cause; the tunics of the vessel, weakened by the structural alterations, yield more and more under the force of the blood-pressure. To this view, which is generally accepted, is opposed the important fact that, while aneurism is most usual between thirty and forty, atheroma rarely sets in until after forty. On the other hand, it may be alleged that aneurism would be vastly more frequent if the changes in the structure of arteries occurred earlier in life; and, furthermore, in cases of aneurism, the existence of atheromatous degeneration can almost always be ascertained.

Pathological Anatomy.—In Sibson's* collection of cases of aneurism occupying some part of the aorta, 880 in number, 703 were of the thoracic aorta, the others of the abdominal and its branches. Of these, 193 were of the ascending aorta, 87 occurring at the sinuses of Valsalva. This statistical fact is a confirmation of the pathological law that those parts most subject to strain in the ordinary course of functional work soonest become diseased. Next to the ascending part, comes the arch which was the seat of aneurism in 120, while only 72 were in the descending aorta. As regards the form assumed by the aneurism, two thirds of those affecting the ascending part are examples of the sacculated variety. It is a curious fact that, while aneurisms of either the ascending or transverse aorta are sacculated, those involving both parts of the vessel are cylindrical or fusiform (Sibson). In the descending aorta, the sacculated are about two thirds of the whole number. The direction taken by the aneurism of the ascending aorta is usually to the right of the transverse part, about one half toward the back, the other half to the right and front; of the descending, to the left and posteriorly.

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* Sibson's "Medical Anatomy," London, 1869 (see columns 57–60).

The sac of the aneurism, which in the beginning is composed of the tunics of the vessel, or of the adventitia, is subjected to various pathological influences which alter its character. It is affected by atheroma, by calcification, but is still more changed in structure by attacks of inflammation which unite it to neighboring organs. The author has met with a case in which the proper sac had disappeared, and the walls were made up for the most part of the tissue of the left lung in which it was imbedded. The interior of the sac is altered by successive deposits of fibrin, differing in age, color, and density, and having a distinctly stratified arrangement. The oldest layers are grayish-white, tough, and firmly adherent to the inner surface of the sac, while the recent coagula contain more or less coloring matter, are softer, easily broken up and detached. By the gradual addition of layers of fibrin the sac is ultimately closed, and a cure is effected by the obliteration of the cavity. Sometimes the outermost layers of fibrin undergo calcification; sometimes an acute inflammation is set up and the sac is destroyed by suppuration. Occasionally blood-clots or masses of fibrin are cast off, with the effect to block the efferent vessel, or some of its tributaries, or, breaking up, are distributed as multiple emboli. The mischief caused by an aneurism is not limited to the sac itself, but involves neighboring organs by pressure, interfering with functions, or inducing inflammation, ulceration, and atrophy. The bronchi, œsophagus, or thoracic duct, may be opened by ulceration, or the vena cava occluded by a thrombus, or invaded by ulceration, thus producing an aneurismal varix, or atrophy of the neighboring lung may be caused by pressure. The ribs, sternum, and vertebræ may be eroded, and the spinal cord compressed. Important nerve-trunks are first irritated by the proximity of the tumor, next inflamed by pressure, and ultimately so mixed in the elements of the sac as to disappear. If the aneurism occur in the sinuses of Valsalva, the aortic valves become incompetent by reason of changes in the orifice. It had been generally maintained that aneurism of the aorta causes hypertrophy of the heart, but Sir Dominic Corrigan, Professor Axel Key,* of Stockholm, and others, have shown that "aneurism has no tendency to produce enlargement of the heart" (Corrigan); and, when hypertrophy coexists with aneurism, there is no causal connection.

Termination by rupture is the most common. As regards aneurisms of the sinuses of Valsalva, about eighty per cent. terminated by rupture; of the ascending aorta, fifty-seven per cent. ended by rupture; of the transverse, thirty-seven per cent.; of the descending aorta, seventy-five per cent. (Sibson). Rupture of the ascending aorta occurs into the pericardium (in one half of the cases), into the right auricle, into the lung, into the pleura, into the right bronchus, into the trachea, into the œsophagus, or externally; of the transverse portion, into the trachea, lungs, œsophagus, pleura, posterior mediastinum, pulmonary artery, or vena cava; of the descending portion, into the pleura, lungs, etc.

Symptoms.—The signs and symptoms of aneurism, as of cardiac diseases, are comprehended in two groups: rational and physical. The rational signs are symptomatic of the functional troubles caused by the aneurism, and, of course, vary somewhat with the position of the new formation. It will conduce to clearness to consider the subject of aneurism of the thoracic aorta and its main branches first, and follow with aneurism of the abdominal aorta and its main branches.

Aneurism of the Thoracic Aorta.—The earliest symptom is pain. This may be a fixed pain, almost constant, and felt in one spot under the sternum and in the neighborhood of the aneurism. More frequently the pain has a combined lancinating and tensive character, shooting up from the interior of the chest to the neck, to the shoulder, down the arm to the elbow, sometimes to both sides; or, it is felt in the back and shoots around the chest in the direction of the intercostal nerves. At times the attacks of pain are most severe, and demand the use of active anodynes. These pains, which occupy the trajectory of the cervical and brachial plexus, and of the intercostal nerves, ought not to be confounded with attacks simulating closely angina pectoris, which occur when the aneurism is near the heart. These paroxysms consist of præcordial pain and anxiety—pain shooting across the chest, in the præcordial region, and to the shoulder, down the arm. Although these attacks are due to the irritation of the nerve-trunks, they affect a different set of nerves, those supplying the heart itself. So constant is this symptom of pain, so severe and persistent, although paroxys-

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* The "Medical Times and Gazette," June 4, 1870.

mal, that, if it come on in a man of middle age without any explanation, aneurism should be suspected in the absence of more characteristic symptoms. There is also more or less dyspnœa, paroxysmal rather, in the initial period, and may occur without any apparent cause, from pressure on the pneumogastric when there is apt to be nausea associated with it, or to pressure on the phrenic, when there may be hiccough. In the further development of the aneurism, dyspnœa may be produced by pressure on the left primary bronchus, diminishing the air passing to the left lung or on the trachea, or to pressure interfering with the return of blood from the lung, and there may be simultaneously pressure on the pneumogastric, causing laryngeal symptoms, and on the phrenic, causing paralysis of the diaphragm. When the dyspnœa is due to pressure on the recurrent laryngeal, there will be associated with it peculiarities of the voice, cough, and breathing. When due to pressure on the trachea, it is somewhat relieved by inclining the head forward ; and in one case, that of a physician seen by the author, a violent suffocative attack was brought on by raising the head erect. In other cases of pressure on either bronchus, relief to the breathing is afforded by turning to the opposite side. When the dyspnœa is due to direct pressure on the lung, there are present fever, profuse expectoration, etc., the signs of phthisis. When the aneurism is at the arch and springs from the inferior segment, pressure on the recurrent laryngeal will produce characteristic symptoms at an early period. If the pressure irritates without destroying the nerve, all of the muscles of the larynx innervated by it will be thrown into a state of spasm, with the effect to modify the voice and cough in a most characteristic manner. While one cord approximates its fellow and vibrates in the normal manner, the other is in a state of rigidity and does not vibrate normally, producing an odd effect on the voice, there being a double tone, one high-pitched and the other lower ; but this *vox anserina* occurs with both inspiration and expiration. Alteration of the voice is much more common than aphonia. When the paralysis of the vocal cords is double, which is an extremely rare event, the voice is gone and there is aphonia ; but, if, as is usually the case, the paralysis is of the left vocal cord, the voice has a harsh, stridulous character. The cough exhibits the same peculiarities. When the nerve is irritated without being destroyed, the cough is loud, resonant, and metallic—croup-like ; on the other hand, when the nerve is destroyed and the muscles of the larynx paralyzed, the cough is suppressed, wheezy, stridulous. By laryngoscopic examination, the explanation of these phenomena is afforded in the character of the movements of the arytenoid cartilages and vocal cords. The effect of irritation is seen in the rigid state of one cord, which does not approximate accurately its fellow during phonation, and vibrates imperfectly if at all. When the destruction of the nerve is effected and paralysis comes on, the paralyzed sure, and ultimately so mixed in the elements of the sac as to disappear. If the aneurism occur in the sinuses of Valsalva, the aortic valves become incompetent by reason of changes in the orifice. It had been generally maintained that aneurism of the aorta causes hypertrophy of the heart, but Sir Dominic Corrigan, Professor Axel Key,* of Stockholm, and others, have shown that "aneurism has no tendency to produce enlargement of the heart" (Corrigan); and, when hypertrophy coexists with aneurism, there is no causal connection.

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The state of the pupil has a high degree of clinical importance. If the aneurism irritate the fibers of the sympathetic nerve without destroying them, this fact is signalized by permanent dilatation of the pupil ; but if the nerve-fibers are destroyed, paralysis of the radiating fibers of the iris ensues, and hence contraction of the pupil follows (the third pair unopposed). Usually spasm of the glottis (irritation of the inferior laryngeal) coincides with dilatation of the pupil (irritation of the sympathetic) ; but this relation is not invariable, for spasm of the glottis may be present with contracted pupil (Russell). Unilateral sweating of the head and face is a symptom which occurs in a small proportion of cases, and may or may not be coincident with changes in the pupil. The sweating is strictly limited to one side of the head and face, and, although increased by external warmth and exercise, comes on quite independently of external conditions. It is supposed to indicate irritation of the sympathetic, but the real nature of the phenomenon is as yet unknown. As unilateral sweating is produced by a variety of causes, it is of importance in this connection only when it coincides with other and more definite signs.

The character of the cough associated with laryngeal troubles has been mentioned. There is also cough when the lungs are involved, and sometimes profuse expectoration. Cough is a symptom of pressure on the trachea or bronchi. Expectoration of blood from a minute communication between the sac of the aneurism and trachea is one of the puzzling symptoms, for it may have all the characteristics of an ordinary pulmonary hæmorrhage. This escape of blood may continue for several weeks by a circuitous channel, before rupture finally occurs. Dysphagia or difficulty of swallowing is produced by the same mechanism as the laryngeal spasms : irritation of the pneumogastric is reflected over the motor branches distributed to the œsophagus. This does not continue a permanent disability, but persists for a few hours, then disappears, to return again at some uncertain period. Pressure of the aneurism on the œsophagus produces a more permanent dysphagia, and, as might be expected, is a more common symptom in aneurism of the descending aorta than in any other position. According to the statistics of Sibson, dysphagia was present in thirty-five per cent. of cases of the descending aorta, in thirty-one per cent. of those of the arch, and in only two per cent. of those of the ascending aorta. As the aneurism enlarges, important symptoms are produced by pressure on the great vessels. If the descending cava is obstructed, bilateral œdema of the face and arms follows, or, if the innominata only is compressed, the effusion is limited to the right side or to the left side, according as it is the right or left vein. When the right auricle is impinged on, there must ensue cyanosis, general venous stasis, and dropsy ; when the left auricle, pulmonary congestion with its consequences—brown-red indurations, hæmorrhagic infarctions, etc. Dilatation of the lymphatic vessels will be produced by the pressure of an aneurism occupying the last portion of the arch and the descending aorta.

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* "The British Medical Journal," December 19, 1874.

When an aneurismal tumor protrudes at the thoracic wall, the diagnosis by the physical method becomes much simplified. By palpation, the existence of a tumor, pulsating and swelling with each pulsation, is made out. The first beat is stronger and more prolonged than the second, if there are two, and is a little subsequent to the heart-beat, while it anticipates the radial pulse. The second corresponds to the diastole of the heart, and is the recoil from the closure of the aortic valves, and of course is indistinct or wanting when the aortic valves are incompetent. A double pulsation exists only in the case of recent aneurism, and of the thoracic aorta ; old aneurisms, lined with thick layers of fibrin, or composed of bony tissue, can not be thrown into vibration by the comparatively feeble force of the recoil wave, and abdominal aneurisms lie at too great a distance. Palpation also reveals a peculiar thrill or tremor which is intermittent, or is synchronous with the first beat, and is known as *aneurismal thrill.* It is obvious that, to feel this, a tumor must be very superficial, and without dense, thick, or bony walls. In the case of aneurisms deeply placed in the thoracic cavity, these symptoms ascertainable by palpation are wanting. Dullness on percussion is elicited only when the aneurism has attained sufficient size or is in a position to cause the reaction, and it exists over a very limited area under any circumstances. The usual position of the dullness is on the right of the sternum, parallel with the second or third rib ; or it is at the sternum, or to the left of the sternum, and posteriorly to the left of the spinal column. This symptom does not afford precise indications, since the dullness of aneurism does not differ from that caused by any tumor, or by a solid organ, or by a purulent depot. On auscultation we hear in aneurism a systolic and diastolic sound or shock, such as is audible over the artery itself. These sounds correspond to the pulsations, with the exception, however, that a diastolic sound may occur when there is a systolic and not a diastolic pulsation. The mechanism of their production is obvious enough, the systolic sound being due to the vibration of the column of blood propelled into the sac, and the diastolic to the recoil from the shutting of the aortic valves. The second or diastolic sound has a "booming" quality, and is heard the more perfectly the nearer the heart the aneurism is placed. When there are cardiac murmurs of stenosis or insufficiency, or peculiarities of accentuation, they are propagated to and are audible over the aneurism. The fitness of the expression, that when aneurism is present "two hearts are beating in the chest," is quite obvious; so close, indeed, is the resemblance that the sounds heard in aneurism were considered by Laennec as cardiac entirely. Murmurs also occur in aneurism with, or take the place of, the sounds; they are formed in or of the sac, and are not propagated from the heart. They are by no means common, and a diastolic murmur is greatly less frequent than a systolic. They are produced by some irregularity in the interior of the sac, or by pressure on a neighboring vessel, or on an adjacent part of the aorta. A sacculated aneurism does not, but the other varieties do in some cases, retard the pulse-beat. If it occupy the ascending aorta the pulse will be behind on the whole round of the circulation; if the transverse portion of the arch and between the arteria innominata and the left subclavian, the pulse of the radial will be retarded; if the descending aorta, the femoral pulse will be delayed. The pulse is also changed in character. If the orifice of the efferent vessel is unobstructed, the normal dicrotism of the pulse is increased because of the secondary undulation imparted to the blood-column; on the other hand, if the efferent vessel is narrow or obstructed, the pulse is small, irregular, and without dicrotism.

The symptoms of aortic aneurism vary with the position of the sac in the course of the vessel. In aneurism of the ascending part there are pressure on the right auricle, cyanosis, venous stasis, and dropsy. The aortic valves are usually incompetent, and the murmurs thus produced are audible over the sac. As the tumor develops anteriorly, the pulsation is felt in the second or third right intercostal space at the border of the sternum. When it projects it forms an hemispherical tumor, having, usually, a double pulsation, a reddish and purplish tint, is crossed by enlarged and varicose veins, and presently softens. The radial pulse is retarded equally on both sides, unless compression of the innominate artery occurs. The laryngeal symptoms, so constant in aneurism of the arch, are wanting, but the pupillary phenomena and the unilateral sweating may be present. The trachea and œsophagus are occasionally encroached upon, but the right primary bronchus may be compressed. In about one half of the cases the pulmonary artery and the adjacent right ventricle are impinged on. According to the data of Sibson, aneurisms of the ascending aorta compressed the right lung in thirty-four instances, the left lung in ten, the right bronchus in six, the left bronchus in one, the pulmonary artery in seven, the descending vena cava in sixteen, and the trachea and œsophagus in nine each. In aneurism of the arch there will be œdema of the head and upper extremities; the pupil will be affected but not invariably; laryngeal symptoms will be usually present from compression of the left recurrent nerve; there will be compression of the left primary bronchus, and consequent feeble respiration or collapse of the left lung; there will be dysphagia from obstruction of the œsophagus sometimes; attacks of angina pectoris from irritation of cardiac nerves. Referring again to the facts of Sibson, we find in regard to aneurism involving both the ascending and transverse aorta, that there were present dyspnœa in 74 per cent., orthopnœa in 21·5, cough in 47, hæmoptysis in 10, stridulous breathing or affection of voice in 17, dysphagia in 21·5, the head and neck were swollen in 14 per cent.; while in aneurism of the transverse aorta alone there were present, dyspnœa in 71 per cent., orthopnœa in 20 per cent., cough in 57·5 per cent., hæmoptysis in 19 per cent., inspiration stridulous in 47·5 per cent., dysphagia in 31 per cent., the pulse weaker in one wrist in 26 per cent. As regards the descending part of the arch of the aorta, we find that the vertebræ were eroded in 42 per cent.; the tumor made pressure on the trachea in 12·5 per cent., on the left primary bronchus in 37·5 per cent., on the œsophagus in 31 per cent., the left lung in 48 per cent.; dyspnœa occurred in 50 per cent., cough in 46 per cent., the voice affected in 25 per cent., and dysphagia existed in 33 per cent. The important disturbances arising from aneurism in this situation are obviously due to the recurrent laryngeal nerve, left primary bronchus, œsophagus, and trachea, which come into close relation with the vessel at this point. Aneurisms lower down compress the left lung, and cause erosion of the vertebræ in 74 per cent. There is a fixed boring pain about the site of the aneurism in one half the cases; there is also much pain in the intercostal nerves; the femoral pulse is retarded; and, when the spinal canal is invaded, disorders of sensation and of motility occur in the lower limbs, terminating in hemiplegia. A case is reported of an aneurism of the arch, dissecting downward between the trachea and œsophagus and bursting into the stomach. The symptoms were orthopnœa, dysphagia, and stricture of the œsophagus, but not of aneurism.*

Aneurism of the innominata causes very much the same symptoms as the first part of the arch: a systolic and a diastolic pulsation; a double sound, synchronous with the cardiac, and audible with the greatest intensity at the junction of the clavicle and sternum; retardation and increased dicrotism of the right radial pulse if unobstructed at orifice of exit; pain in the neck and arm; compression of the descending vena cava, and œdema of the head and upper extremities, or there may be compression of the left vena innominata, and consequent œdema of the left side of the head and the left arm.

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* "Pathological Society's Transactions," vol. xxvii, p. 97, report of Dr. Frederick Taylor.

Aneurism of the Abdominal Aorta.—The point of election is at or near the cœliac axis. In Dr. Sibson's collection of cases, 177 in number, 131 occurred at this point. Less than one half arise from the anterior face of the vessel, and consequently the vertebræ are eroded in a large proportion of cases—55 per cent. The variety of the aneurism is the so-called false, and the form sacculated in 60 per cent., and they attain considerable size, sometimes to a capacity of ten pounds.

Aneurism of the abdominal aorta is usually referred to a violent muscular effort—always, in the author's experience. It appears to be less associated with atheromatous degeneration of the arteries than is aneurism of the thoracic aorta. One of the earliest symptoms is pain, felt in the position of the tumor and radiating through the abdomen. As the aneurism is so situated that the semilunar ganglion and the nerves of the solar plexus must be compressed by it, pain is necessarily produced, and, as the nerves radiate from a common center, the pain also radiates, shooting up into the hypochondria and downward to the iliac regions and hypogastrium. These pains are paroxysmal, and may disappear for hours and days; but the attacks are of extreme severity, and when they subside leave the patient exhausted. The local pain seems to the patient to be in the stomach, and, as this organ is disturbed in function also, the attacks are often confounded with gastralgia. This local pain is more constant than the other, and there is rarely an entire cessation of it, although it may be little more at times than an uneasiness. In about one half of the cases the most violent pains occur in the back, and shoot down through the lumbar region into the hips along the course of the sciatic nerves. There is here also a fixed, boring pain felt opposite the cœliac axis, which is rarely absent. In both situations the pains are aggravated by pressure, by sudden jolting, or bending the body. The pain in front is increased by taking food, especially by distention of the stomach. Distress produced by eating, indigestion, flatulence, and nausea, are early symptoms, due to irritation of the solar plexus. As the pain is brought on by eating, and as pronounced stomach troubles are present in a majority of the cases, it need occasion no surprise that they are often supposed to be entirely stomachal. This mistake is persisted in even when a tumor is present, and the phenomena are then ascribed to cancer of the stomach. This mistake is all the more readily made, since the interference with digestion brings on a cachectic state with wasting, and since jaundice may be caused by pressure on the common duct. The stomachal disorders are less pronounced in those aneurisms springing from the posterior part of the aorta and making their way posteriorly. According to Sibson, a pulsating tumor was observed in 55 per cent. of the cases. A large tumor may form posteriorly, and produce extensive erosions of the vertebræ, without being ascertained by the most careful palpation. A dislocated kidney, a migrating spleen, a bunch of enlarged lymphatics, may rest on the aorta and receive a pulsation synchronous with the cardiac systole. In applying the method of palpation, to determine the nature of a pulsating epigastric tumor, the sources of error just mentioned must be eliminated by putting the patient in such a position that these bodies will fall away from the aorta, when, of course, the pulsation will cease. The aneurismal tumor is situated usually in the epigastrium, a little to the left of the median line. It is a globular, elastic tumor, pulsating with an expansile movement in all directions, and on inspection there will be seen a swell of the whole abdomen with each pulsation. The pulsation of an abdominal aneurism is single, a little later than the cardiac systole, and there is usually a thrill. If pressure is made on the aorta below the aneurism, the sac will be filled with a stronger impulse, and retain its fullness, while the thrill ceases or is less marked. Percussion is of little value. Dullness may be elicited under favorable circumstances, but this affords no indication of the nature of the producing cause. Murmur is present in a considerable proportion of cases. It has a blowing character, is rather soft, and, in time, is a little later than the cardiac systole. When the aneurism springs from the anterior surface of the aorta, the murmur is audible in front, and, when the growth is posterior, audible behind; rarely is it audible in both situations in the same case. Standing erect arrests the murmur, because, according to Corrigan, of the increased tension in the sac produced by the superincumbent column of blood. To this statement and explanation must be opposed the important fact that the murmur was audible in the erect and ceased in the recumbent posture in an undoubted case of aneurism. Aneurism of branches of the aorta are occasionally encountered. An aneurism of the mesenteric artery is a movable tumor which may be confounded with floating kidney.* It differs from the latter in being globular and pulsating. Aneurism of the hepatic artery may cause jaundice, by pressure on the duct, or ascites, by pressure on the portal vein. As they are small in size and deeply placed, aneurisms of the hepatic artery are rarely, if ever, recognized during the life of the individuals affected by them.

Course, Duration, and Termination of Aneurisms of the Aorta.— The course of aneurism is much influenced by the condition of organs compressed, and the disturbances of function thus induced. They are essentially chronic, slow in development usually until of sufficient size to compress the organs about them, when symptoms are caused which attract attention to them. Not all cases give rise to symptoms that indicate the cause of the disturbances which they produce; only the disturbances are recognized and treated as the real malady. Thus, aneurisms deeply placed in the thorax posteriorly, or of the abdominal aorta, high upon between the crura of the diaphragm, or growing toward the lumbar region, may produce no symptoms which can indicate the nature of the disease. Even when a tumor of considerable size exists, in the situation most favorable for recognition, grave doubts may be entertained as to its aneurismal character. They may terminate in a variety of modes; by exhaustion, by pneumonia, by rupture and hæmorrhage. Probably the most useful collection of statistics showing the course and terminations of aneurism is that of Sibson, and the author prefers, therefore, to illustrate these points from it. As regards aneurism of the first part of the aorta (sinuses of Valsalva), we find that 80 per cent. terminated by rupture, 45 per cent. into the sac of the pericardium, 13·5 per cent. into the pulmonary artery, 8·5 per cent. into the right auricle, 5 per cent. into the right ventricle, and 5 per cent. into the left ventricle. Aneurism of the ascending aorta "ruptured in 57 per cent.; externally in 8, into the pericardium in 22, into the pulmonary artery in 4, into the descending vena cava in 5, into the right lung in 5, into the left pleura in 4, "etc. In a series of 25 cases published in the "New York Pathological Transactions,"* the termination was by rupture; and in almost all of the cases death occurred suddenly, but few of them having been diagnosticated. Aneurisms of the ascending aorta and arch conjointly ruptured in 37 per cent., into the pericardium in 10, into the vena cava 4, into the trachea 4, etc. Aneurism of the descending part of the arch ruptured in 75 per cent., into the trachea in 4, into the left bronchus in 16·5, into the left pleura in 23, into the right pleura in 12·5, etc. Aneurism of the abdominal aorta ruptured in 77 per cent., into the peritoneal cavity in 28·5 per cent., into the subperitoneal tissue, in the left hypochondriac region, 22 per cent., etc. Although death is almost immediate when an aneurism ruptures, yet this is not invariably the case. A small opening may exist in the trachea, permitting a little blood to escape from time to time, simulating pulmonary hæmorrhage, and continuing to discharge in this way until a complete rupture occurred at the end of several months. These are called "weeping aneurisms." Gairdner† records a case of this kind in which the opening was blocked by some fibrin, and continued so for four years. An opening externally may discharge slowly, of which notable examples have been published—a free and fatal hæmorrhage being prevented usually by a plug of fibrin. As the beginning of an aneurism is very uncertain, it is difficult to state its duration within exact limits. They vary exceedingly in duration; from fifteen days to thirty years are the extremes which have fallen under the author's notice. Much depends on the influences, medicinal and moral, to which the patient is subjected. Some cures are effected.

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* Dr. Burney-Yeo communicates a case to the Pathological Society ("Transactions," vol. xxviii, 1877), in which the first part of the artery was affected and not movable. It compressed *both* renal arteries, and caused death by uræmia.

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* Tabulated in "Transactions of the London Pathological Society," vol. xxix.

† "Clinical Medicine," *op. cit.*

Prognosis.—Aneurism must be regarded as a very grave disease. Under the improved methods of medical treatment now available, more cures are effected than formerly, and the question of treatment must enter largely into prognosis. Under any circumstances, a qualified opinion only should be given, for an aneurism that is apparently solidifying may take an unfavorable turn, and death be caused by some intercurrent malady.

Treatment.—The object of the medical treatment of aneurism is to secure the solidification of the sac. As this has occurred several times spontaneously, without the intervention of art, it is more difficult to assign to remedies their exact share in any successful treatment. To obtain coagulation of the blood in the sac and to effect the solidification of the fibrin are the objects before us. If we have to deal with a sacculated aneurism, the closure of the sac can be accomplished without interrupting the current through its proper channel. The importance of this is very obvious in dealing with the aorta, for no collateral circulation is here possible. The difficulty of a case is immensely increased from the therapeutical standpoint, when we have to treat a dilated vessel. The treatment by rest, as absolute as can be maintained, is a very old method, and has much to recommend it even now. If the patient maintains a position of recumbency, and moves in that position as little as possible, the action of the heart is slowed and its force lessened, so that the blood in the sac may coagulate. Formerly, the abstraction of blood and an absolute diet were combined with rest in the recumbent posture (Valsalva's plan), but, in the more recent method of Tufnell, only the rest and a restricted diet are considered necessary. The diet of this plan consists of two ounces of liquid and four of solid food morning and evening, and four ounces of liquid and six ounces of solid at mid-day.* In addition to this restricted diet, the blood-pressure is reduced by the daily use of laxatives. The period of confinement to a recumbent posture is from eight to thirteen weeks. The results obtained by Mr. Tufnell are certainly very satisfactory, for he has reported cases of aneurism of the abdominal aorta solidified in thirty-seven and twenty-one days, and one of popliteal cured in twelve days; and he affirms that, "if the plan of treatment by position be but *steadily* and *perseveringly* carried out, a successful issue can (in suitable cases) almost be guaranteed." In addition to rest, arterial sedatives are sometimes given, with the view to keep the action of the heart still lower than that rate of movement attainable by rest merely, according to Tufnell's plan. The arterial sedative employed for this purpose is the tincture of veratrum viride, given to bring down and to keep the pulsations about fifty per minute. The author has witnessed successes obtained in this way. Bloodletting is admissible in cases of large aneurism, a rupture being threatened by violent action and plethora. Recently, important results have been obtained by the free administration of the iodide of potassium, or *iodide of sodium*, which acts in the same way, but is less hurtful (gr. xv.—℈j) three times a day. It has a remarkable influence over the pain, probably because of its effect in diminishing the tension of the sac, the force of the heart, and the blood-pressure (Balfour). Besides this, the iodide seems to affect the sac itself. The use of the iodide of potassium may be combined with rest and a lowered diet, but these are only adjuvants, and are not essential to the treatment.

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* "Medico-Chirurgical Transactions," vol. xxxix, 1874, p. 83, *et seq.*

Since the publication of the last edition of this work, much new testimony has been published, especially by our French colleagues—Huchard, See, and others—in regard to the remarkable powers of the iodides for bringing about slow structural alterations of connective tissue, and of granulation tissue. In chronic endarteritis, the sclerosis can be removed by peristent use of the iodides, and of these they find that sodium iodide can be given the requisite time, and in the necessary quantity, to accomplish the object without hurt to the system. Not a little incredulity has been expressed on this side, but these adverse opinions are not based on any proper investigation; and on the other hand some of our best therapeutists have long entertained the same opinions.

Langenbeck has called attention to the great value of ergot as a remedy in aneurism, and has reported some successful cases. It has been used since with advantage. Its employment is based on the action which it exerts on the muscular fiber of the arteries, and, therefore, it is asserted, it can have no effect on the aorta. Those who use this argument forget that ergot slows the heart, and raises the blood-pressure at the periphery by contracting the arterioles—conditions highly favorable to promote coagulation of the blood in the sac. Two to five grains of the so-called ergotin, which is the aqueous extract,[*] should be administered hypodermatically, simply dissolved in water and filtered. This practice may be continued while the other measures are being carried out, as there is no therapeutical incompatibility. The success which has lately been obtained with barium, based on the experimental research of Boehm, is a beautiful example of the value of such investigations. From two to five minims of the liquor barii chloridi, well diluted, or one sixth grain to one grain of the salt in pill form, may be given three times a day, after meals. The physiological effects of this medicine on the vessels suggested its employment originally. Acetate of lead also affects the vessels—especially the muscular layer—but there are very obvious objections to its long-continued use.

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[*] Squibb's preparation is probably best.

Attempts have been made by direct means to secure the coagulation of blood in the aneurismal sac. These consist in the introduction of fine wires, horse-hair, etc., with the intent to supply a foreign body about which the blood will coagulate. Thus far, these attempts have been failures, except one success reported lately. Another method, of which very confident expectations were at one time entertained, is the method of *electrolysis*. This consists in the introduction of an insulated needle into the interior of a sac, and the application of a sponge electrode to the exterior, through which a galvanic current is passed. The blood coagulates about the needle. Much discussion has resulted as to the pole, anode, or cathode, to be introduced into the sac. As about the positive pole acids, oxygen, etc., collect, a firmer clot is there formed; while about the negative, hydrogen and the alkalies, producing a softer clot. The positive electrode needle is withdrawn with difficulty from the sac, owing to the firmness and adhesiveness of the adherent coagulum, and in making the effort there is danger of hæmorrhage and of setting free multiple emboli. On the other hand, although the clot produced by the negative needle is less firm, it acts as a nucleus about which denser coagula will form afterward. Although cures have been reported by electrolysis, this method is not so successful as others recommended above. Furthermore, the danger of hæmorrhage, of exciting inflammation, of detaching large clots in the circulation, is so great that this plan is not to be commended.

Aneurism of the coronary artery is a rare disease. Crisp* has collected and tabulated twelve cases. They occurred from eleven to seventy-seven years of age, but chiefly after forty, and in subjects exposed to such injury by occupation. They may cause sudden death without symptoms, or there may be suffocative attacks, pain, and palpitations. They vary in size from a pea to a walnut, and rupture into the pericardium. This is not the invariable termination, although usual, death being caused in three of Crisp's cases by bronchitis, exhaustion, and an unknown cause unconnected with the aneurism.

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* "Transactions of the Pathological Society," vol. xxii, p. 108.

DISEASES OF THE RESPIRATORY ORGANS.

TOPOGRAPHY OF THE CHEST: PHYSICAL DIAGNOSIS.

THE regions into which the chest is divided for the purpose of physical exploration are given in Figs. 1 and 2, and the relations of the external lines and divisions to the contents of the cavity are shown in Fig. 13. The student should fix in his mind, especially, the position of the contained organs, with reference to the exterior anatomical landmarks.

The relations of the heart and great vessels have been discussed in the section devoted to diseases of the vascular system. It remains now to determine the relations of the lungs, and so much of the heart and great vessels as may be necessary to a proper understanding of the practice of physical diagnosis in pulmonary diseases.

In making a physical exploration of the chest, the position of the patient should be carefully arranged. In males the chest should be entirely bared, and in females covered with a close-fitting chemise or flannel undershirt. If the condition of the patient will admit of it, the best position for the examination is the erect posture, with the hands folded on the hips behind, when the front of the thorax is to be explored; with the arms folded on the chest, and the body slightly inclined forward, when the back is to be examined.

The examination should be conducted in an orderly manner. It includes *inspection*, *palpation*, *mensuration*, *percussion*, and *auscultation*. Some practical observations on these methods may fitly precede a study of the maladies in which they are applied.

Inspection.—By this term is meant a survey of the exterior of the thorax; of its size, shape, deviations from the normal, of its movements.

The thorax may be contracted either in consequence of original defect, or from disease in early life interfering with the play of the lungs. A very common condition is that known as *pigeon-breast*, the ribs having yielded at the sides, giving the sternum an apparent prominence. The chest of rickety subjects is also comparatively common, the pigeon-breast being one of its features, but there are, also, a deep groove along the line of junction of the cartilages with the ribs, nodosities on the ribs near the cartilages, and flattening of the chest posteriorly, the ribs being rather sharply bent at their angles. By inspection, the greater size of one side; the absence of the proper motion, and filling out of the intercostal spaces, or the opposite condition of retraction; bulging at special areas; retraction of the intercostal space at the point of apex-beat, etc., are alterations to be observed.

By inspection may be noted whether the respiratory movements are rhythmical, thoracic, or abdominal. In children and males the respiration is largely abdominal; in females, thoracic. The breathing may have the Cheyne-Stokes type; i. e., beginning with movements normal in number, the rate of breathing lessens, and the depth and force diminish progressively, until at length breathing seems to be suspended, the face becomes cyanosed, consciousness appears to be lost—then, with a start, respiration is resumed with increasing frequency until the maximum is reached, when again the number of respirations declines to zero.

In beginning an examination of the chest, an attentive inspection should be made of the whole thorax during the movements of inspiration and expiration.

Mensuration.—For ordinary purposes, a common tape-measure suffices to ascertain the size of the thorax, of its lateral halves, and of special parts deviating from the normal. The circumference of the chest is taken at the level of the nipples, and the difference between the completest expiration and the fullest inspiration is known as the *expansive mobility*. This difference should be about three inches, but practice by developing the chest muscles may greatly increase the mobility. No adult chest is normal the expansive mobility of which is less than two inches.

Palpation.—Important information is had from palpation, which consists in applying the palmar surface of the hands to the chest, and noting the changes, if any, in the condition of the parts. When the fingers are applied to the intercostal spaces, the degree of resistance becomes an element of diagnosis. Resistance is increased by anything causing induration of pleura or lungs. When the intercostal spaces are distended, as by hydrothorax, or the lung is solidified, the character of the resistance is changed from the peculiar elastic quality belonging to the parts in a normal state.

The vibration imparted to the chest-wall by the voice, by the friction of inflamed pleural surfaces, and by the movement of mucus in the larger tubes, is called *fremitus*. *Vocal fremitus* is produced by the voice, as when the patient says in a distinct tone "twenty-one," or "ninety-nine," or any similar words. That is *tussive fremitus* when caused by cough, and *friction fremitus* when the rubbing together of inflamed pleural membranes imparts a trembling or vibration to the chest-wall. To ascertain it, the hands are placed flat on the chest at various points, according to the situation of any morbid process in the thorax. The rhythm of the vibration is a point to be noted; as, for example, when a pleural friction fremitus is to be distinguished from a pericardial. Vocal fremitus is increased by any cause increasing the density of the pulmonary substance, as in pneumonia, phthisis, etc.; it is lessened by any cause separating the lung from the chest-wall. It must be remembered that normally the vocal fremitus is greater on the right than the left side, especially over the primary bronchus.

Percussion.—Certain notes are produced by percussion of the walls of the chest. The mode of eliciting them is *mediate* or *immediate;* by the former, a body is interposed—a *plessimeter*—on which the blow is struck; by the latter, the percussion is made on the chest-wall. Plessimeters are made of bone, ivory, or hard rubber, and some of them have a scale to mark the dimensions of the part percussed. The most convenient plessimeter is the finger, being of material having the same physical qualities as the parts percussed, and adapting itself perfectly to the surface. If any air is interposed between the plessimeter and the surface beneath, the percussion note has a clacking, metallic quality.

The hammer usually employed is composed of the three fingers of the right hand, so arranged that their extremities are on the same plane. In very delicate percussion, a single finger may be used.

The hammers which I find most useful are of several sizes, and of differing weight. The most frequently used nowadays is of rubber, conical in shape, and attached near the middle to a metal handle tipped with ivory or bone or celluloid. The hammers having this shape vary in weight, and are adapted to light or strong percussion. The heaviest of these now made are not of sufficient weight for all purposes. A metal hammer, with the percussion end composed of a rubber tip, is at present a popular instrument, and, as this can be made of any size or of any suitable configuration, it will continue to be employed. Where heavy percussion is necessary, I have used with much satisfaction a triangular rubber hammer, the larger extremity being employed for developing the "knee-jerk" and for other purposes, while the smaller end can be used for percussion of any part. The weight of the larger extremity of the hammer gives a firmness to the stroke, although it may be directed with the utmost gentleness. In the other hammers, for the most part, the rubber tips with which they are provided are too extensile, and hence the density of the rubber is increased at the point of impact, and varies from its usual state.

Plessimeters of hard rubber, having a transverse bar for fitting intercostal and other spaces and another bar on which the percussion blow is made, are now in use. They answer an admirable purpose, if the form is such as to permit exact adaptation to the part where the percussion is done. To be entirely successful, each plessimeter should have a projecting point which can serve as a handle.

Formerly I was much opposed to any other hammer or plessimeter than the fingers, but enlarging experience has taught me that rather better results can be had with the instruments described above.

On percussion of the normal chest, a characteristic note is obtained, which becomes the standard of comparison for all sounds produced by this method. It may be entitled the *normal pulmonary percussion sound.* This normal resonance may be variously altered: it may be *increased* or *exaggerated,* or it may be *tympanitic* in quality.

The resonance may be reduced below the normal: the percussion note may be simply *dull,* or it may be entirely *flat.* When percussion is made over a hollow organ, as the trachea, or over a cavity, the note is said to be *tubular.* When the cavity contains air, also, the resonance has a metallic and hollow character, and is then known as *amphoric.* A higher-pitched sound, still more metallic in quality, is called the *metallic.* Another percussion-note, similar in quality to the last, is the *cracked-pot sound,* "*bruit de pot fêlé.*" To produce this sound effectively, percussion must be made during expiration, the patient keeping the mouth open. The sound may be very successfully imitated by forming a hollow cavity with the hands folded, and striking on the knee.

Auscultation is the art of hearing the various audible sounds produced within the cavity of the thorax, in both healthy and morbid states. The ear unassisted can be applied directly to the chest, or the hearing function can be supplemented by an instrument called the *stethoscope.* The utility of such an instrument, and the comparative value of the various kinds of stethoscopes, have been subjects of almost interminable controversy. The majority of physicians prefer the wooden cylinder, but the author, on the other hand, finds that the Cammann's double stethoscope gives the best results in his own hands. The double stethoscope has many advantages; binaural audition, like binocular vision, is more accurate, better defined, and comprehensible than uniaural; the double instrument, by excluding exterior noises, conveys the thoracic sounds to the ear with far greater distinctness, and thus prevents diversion of the attention from them. The manifold sounds of a great city interfere in the most perplexing way with the chest-sounds, and hence the utility of a binaural instrument. The author has found it very useful to employ the unassisted ear, the cylinder stethoscope, and the binaural in every case involving any difficulty or obscurity of the physical signs. The defects of the one mode of observing may be compensated for by the greater clearness or defining power of the other. When the ear is applied directly to the chest, a towel or napkin, only, interposed, the method is entitled *immediate auscultation;* when the stethoscope is used, it becomes *mediate auscultation.*

When the ear is applied to the chest by mediate or immediate auscultation, in health, certain sounds appropriate to certain special situations are heard. The soft, breezy sound produced by the entrance and exit of air from the pulmonary alveoli, is called the respiratory murmur—the *pulmonary or vesicular murmur.* The inspiratory murmur is of longer duration, is louder (higher pitched) than the expiratory, which may be indeed, and often is, inaudible. The passage of air through the larynx and trachea causes a sound of considerable intensity, high pitched, and of large volume. This murmur or sound begins with the act of inspiration and persists throughout it; then an interval of silence coincident with the time between the respiratory movements occurs; and the sound is resumed with, and continues during, the act of expiration. The expiratory portion of the sound is rather higher in pitch, longer in duration, and coarser in quality, than is the inspiratory. This murmur, heard by applying the stethoscope over the trachea, is the *tracheal murmur*, really the *laryngeal*, for it is caused by the movement of air through the larynx.

The *bronchial murmur* is the sound caused by the movement of air through the primary bronchi, or is tracheal in origin but propagated through the bronchi. It is a coarse, blowing murmur, not so high pitched as the tracheal, and is shorter in duration. The interval between inspiration and expiration is comparatively short, nor is it so distinct as that between the tracheal inspiration and expiration.

Intensity.—The breath-sounds may be variously modified *in intensity*. They may be increased or lessened. When the vesicular murmur is simply increased, especially or entirely in inspiration, the change is entitled *puerile*, because of the resemblance to that intensity characteristic of early life. When lessened in intensity, there is simply a diminution in the volume of the respiratory sound communicated to the ear, without any necessary change in quality or duration.

Rhythm.—There are certain changes in *rhythm*. The most significant of these is the so-called Cheyne-Stokes breathing. This is characterized by a deep, prolonged inspiration followed by a succession of regularly diminishing respirations until zero is reached, then a cessation of respiration of less or greater duration, with a deepening cyanosis, and then the resumption of respiration increasing slowly in volume and depth until the maximum is again attained.

Prolonged expiration is a comparatively common alteration of rhythm. As has been stated, in the normal condition of the breathing organs, expiration is comparatively short, feeble, or wholly inaudible; but it may be greatly prolonged, inspiration continuing the same or actually shortening in duration.

The respiration may be jerky or jumping, affecting chiefly inspiration, but expiration may have the same character, especially when abnormally prolonged.

Quality. — The breath-sounds are variously altered in quality. They may be simply harsh, or less soft and breezy than normal. The increased roughness or harshness may be exaggerated into *blowing* respiration, which is similar to *bronchial* breathing, except that it is higher in pitch and clearer than the latter. Bronchial breathing corresponds in quality to the normal sound, and only assumes a pathological character when it becomes audible in unusual situations. *Tubular* breathing is an increased or exaggerated bronchial sound. It is high-pitched, rather metallic and concentrated, and appears to be sounded directly into the ear.

Cavernous, is a rather low-pitched and hollow sound, produced by the vibration of a cavity, and resembles *amphoric*, which is a still more hollow sound with a somewhat metallic quality.

Breath-Sounds—Râles.—Certain adventitious sounds caused by the movement of the air through tubes in a pathological state are entitled *râles* by the French; *rhonchi* by the English; but the former is now almost exclusively used in this country. A râle is a crackling, hissing, whistling, bubbling, or gurgling sound, heard only in inspiration or in expiration, or during both of these acts. Râles may be dry or moist. Dry râles are distinguished as whistling, or *sibilant*, and resounding, or *sonorous*. As the term implies, they are caused by lesions, affecting the diameter of the tubes, without any secretion.

The most important *râles* are those due to the presence of an increased secretion, and the most significant is that râle known as the *crepitant râle.*' This is a fine crackling sound not inaptly compared to the crackling of salt thrown on to bright coals, or still more aptly to the sound caused by rubbing a lock of hair between the fingers in front of the ear, or to the crepitation produced by compression of rubber sponge. The true crepitant râle occurs only with inspiration, is feeble with the first rush of the air inward, and increases in distinctness and volume with a deep and forcible inspiration. Sounds closely resembling the true crepitant râle are that dry crackling which occurs in emphysema, the crepitation of œdema of the lungs, and that which marks the progress of resolution in cases of pneumonia and is called crepitation redux. But this last-mentioned form is larger, more of bubbling in character, and audible often with expiration as well as inspiration.

Subcrepitant, *mucous*, and *submucous râles* are caused by the passage of air through tubes containing fluid. They differ in size, and somewhat in quality. The subcrepitant has a rather crackling character, similar to but larger than the crepitant, and occurring with both inspiration and expiration. The mucous and submucous have more of a bubbling or gurgling character, and are consequently larger and produced in the larger bronchi, as the crepitant are formed in the minuter divisions of the bronchial tree.

Again, certain râles are formed in connection with cavities, dilated bronchi, and cavity-like communications with the pleura. These take name accordingly, and are known as *cavernous*, *amphoric*, *metallic tinkling*, etc.

Voice-Sounds.—The sounds of the voice in speaking, coughing, and whispering are variously modified by disease. *Vocal resonance* may be wanting, or it may be modified or exaggerated. It is wanting when the lung has collapsed and lies against the spine, for example. The most important changes are those of exaggerated resonance. When the voice sounds clearly into the ear, as if coming from a bronchus, when it should, by transmission through the lung-tissue, be much softened, it is called *bronchophony*. The transmission of sounds in a more intense degree, the voice apparently sounding directly into the ear from the chest, is entitled *pectoriloquy*. This modification of voice tone becomes more distinctive by whispering, and is then called *whispering pectoriloquy*. Still another variation of the voice-tone is that entitled *ægophony*. It has a bleating character, a vibratory, nasal tone which is imparted to the sound by a thin stratum of liquid interposed, and is therefore characteristic of pleuritis.

Friction-Sounds.—These are, as a rule, *to-and-fro* sounds, synchronous with inspiration chiefly, and less with expiration, or in the præcordial region, with the cardiac movements. The sound varies in intensity from a mild rustling to a strong, harsh grating. The *creaking-leather sound* is apparently caused by the stretching of old, strong, and ligamentous adhesions, and hence is sometimes audible after the absorption of the liquid exudation of pleuritis.

Succussion.—This is an extremely significant sign, as it can only be produced by the splashing of fluid in a cavity containing air or gas, and hence its value in chest affections. Succussion is effected by smartly shaking the patient from the shoulder. The splashing may be felt by the hand, or heard by the ear on the chest.

The use and significance of the foregoing signs and symptoms will be made apparent in the course of study of the various thoracic diseases.

INFLAMMATION OF THE PLEURA—PLEURITIS.

Definition.—*Pleuritis*, or *pleurisy*, is an inflammation of the pleural membrane. Although not separable by any well-marked signs and symptoms, it is usual to consider two forms, *acute* and *chronic*. It may occur as an independent *primary* affection, or it may be *secondary* to some other disease.

Causes.—There can be little doubt that many cases arise from exposure to cold, especially when a current of cold air is directed against the body in a perspiring state. There is probably a constitutional condition of some kind which determines the seizure, but this state can not be defined. It is more common in early life, up to the middle period, but is uncommon in old age. The secondary disease is much more frequently encountered than the primary. It is very frequently associated with pneumonia, by extension of inflammation through contiguity of tissue ; often, indeed, the pleuritis is the more important of the two affections. It is also associated with catarrhal pneumonia, with bronchitis, pericarditis, embolic pneumonia, pyæmia, abscesses, and other affections of the thoracic organs. It may be excited by caries of a rib, deep-seated (sub-pleural) abscesses, cysts and abscesses of the liver, etc. A dyscrasia may be a cause, when it is said the pleuritis is an intercurrent malady; but it is now known that various morbific matters in the blood may excite serous inflammations, of which rheumatism, gout, Bright's disease, cancer, diabetes, and the eruptive fevers may be taken as examples.

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* "Transactions of the Pathological Society," vol. xxii, p. 108.

Pathological Anatomy.—The initial lesion is hyperæmia of the subserous connective tissue, while red points due to congested vessels are rather thickly scattered over the pleura. Such is the force of the blood-pressure that minute points of extravasation occur on the pleura and in the subserous tissue. The membrane has an arborescent or striated appearance, and is of a reddish or reddish-brown color. The injected portion of the membrane is dull, opaque, and rough; the epithelium is swollen, cloudy, and granular, and is rapidly cast off, while the adherent cells undergo similar changes, and the subserous tissue becomes swollen, infiltrated, and crowded with migrated leucocytes. On the membrane there appears in detached masses, but rather thickly placed, an exudation which makes the surface rough and uneven. Large flakes of exudation may be thrown off, or the membrane may become thickly covered with a more or less heavy coating of fibrinous material. This may also contain a good deal of serous exudation in its meshes, when it presents a gelatinous, felt-like, or spongy appearance. If there be present much liquid, the flakes or masses of fibrin are seen floating in it, or they may be churned up with the serum and form a milky-looking fluid. The exudation which thus forms on the surface passes through various changes. It may undergo fatty metamorphosis, become emulsionized, and disappear by absorption, leaving the membrane unharmed. Adhesions may form by the gluing together of the opposed surfaces, the connecting band of exudation undergoing organization. The membranous exudation on the surface may also become organized; large thin-walled vessels develop from the leucocytes, according to Rindfleisch, and close connections are formed between the neomembrane and the pleura. Again, broad patches of membranous exudation on the opposing surfaces of the pleura uniting by their margins, a central cavity is thus formed in which there may be serum, sanguinolent serum, and flakes of exudation, etc., while close adhesions unite the pleural surfaces all around for a greater or less distance. These secondary cavities form at the base, on the lateral wall of the thorax, and between the pleura and pericardium, and, as they retain the effusion in a fixed position, give rise to errors of diagnosis. Those are examples of *dry pleurisy*, in which a very plastic exudation is thrown out on the two surfaces, over a small extent of the membrane, union taking place, either directly or by a connecting band, there being no other exudation or effusion. It is probable that many of the examples of connecting bands, or adhesions between the pleural surfaces, which are found *post mortem*, no symptoms having occurred during life, were of this character. Usually, however, in pleuritis, a more or less abundant exudation is poured out. According to the nature of the effusion, the cases of pleurisy are divided into the *sero-fibrinous*, the *purulent*, and the *hæmorrhagic*.

In the *sero-fibrinous* form there is poured out from the distended vessels a quantity of fluid, straw-colored and having the qualitative composition of blood-serum. This contains floating in it masses of exudation or flakes, leucocytes, lymph, and red-blood corpuscles, which impart to it a more or less milky or sanguinolent character. The fibrinous part of the exudation consists of layers or folds of whitish, grayish, or reddish albuminous and fibrinous material deposited on the pleura. It may be soft, easily separated, or tough and elastic; and may be readily detached from the membrane, or may adhere with considerable tenacity. When removed, this exudation is found to be closely adherent to a layer beneath, made up of the proliferating connective-tissue corpuscles of the basement membrane, together with a plastic matrix. These layers become ultimately closely connected by the growth of the connective-tissue membrane, or the fibrinous exudation may undergo fatty degeneration and be absorbed. The new connective-tissue membrane, built up as above described, is very rich in vessels, and readily unites with the same formation on the opposing surface of the pleura. The corpuscular elements—leucocytes, lymph-corpuscles, cast-off epithelium, etc.—in the serous fluid may be so abundant as to give it a yellowish or purulent appearance. Hence it may be difficult to make a distinction between this and the truly *purulent form*, in which the serum contains such a quantity of pus-corpuscles that it is thick, yellowish, or greenish yellow. The term *empyema* is applied to a purulent collection in the thoracic cavity. Primary empyema is a very rare event, and, when it does exist, signifies the admission of air or some foreign matter to the cavity. The exudation is at first sero-fibrinous, and becomes purulent, usually not until after the first week. There takes place, under conditions not now understood, a remarkable production of pus-cells—probably by enormously rapid proliferation of the leucocytes which have wandered from the vessels. While the serous fluid has an alkaline reaction, the purulent exudation is acid in reaction. Often the color of the exudation is reddish from the presence of red-blood corpuscles in considerable numbers. But this is not the *hæmorrhagic exudation*, properly. This consists of blood derived from the newly formed, thin-walled vessels of the exudation undergoing organization. A vessel giving way, the blood is poured out (or there is a diapedesis of the red globules) between the layers of the exudation and bursts through into the cavity of the pleura, and, mixing with the serum, forms a bloody fluid. The hæmorrhagic form of pleuritis is usually tubercular in origin, or rather is due to the deposit of miliary tubercle exciting a recurring inflammation. An exudation may be hæmorrhagic when the pleuritis occurs in an individual having the hæmorrhagic diathesis, or who is the subject of purpura.

The evil results of effusions are not limited to the affected membrane. When the quantity is sufficient to displace the neighboring organs, various functional disturbances arise from the compression. At first the lung retracts before the effusion, and only suffers by pressure when the effusion attains a certain volume sufficient to counterbalance its elasticity. As the fluid increases from below upward, the lung at first floats; but gradually the expansibility declines, less and less air enters, and the organ is finally flattened against the spine about its roots. It then appears as a grayish, bluish, or reddish-gray, rather solid and flattened mass, about the size and shape of the adult hand without the fingers. It contains no air, is bloodless, and may be coated with a membranous exudation, or may be bound down by membranous bands. If adhesions exist, the lung will be compressed in part, or, if the organ is infiltrated by caseous or other deposits, the fluid will act on those parts that yet remain compressible. The fluid may be collected in secondary cavities, and compression be confined to those situations. The blood being forced out of the lung, when the organ is flattened against the spine, distends the right cavities, which may dilate, and fills the sound lung, which may become congested and œdematous. If the effusion occupies the right cavity, the heart is forced toward the left side, the diaphragm is pushed down, enlarging the capacity of the right thorax, and displacing the liver downward; if the left cavity, the heart is forced over to the right, the diaphragm is pushed down to a less extent than on the right side, enlarging the left thorax, and displacing the spleen downward. The intercostal muscles become infiltrated, weakened, and, yielding to the pressure, assume a convex instead of a concave shape, the thorax being globular and increased in circumferential and diametrical measurement. If absorption take place and the lung is not adherent, the air will again distend the alveoli, and the thorax assume its normal shape; if the lung can expand again only in part, under the force of the atmospheric pressure, there will take place a depression of the ribs and distortion of the spine to efface the portion of the cavity which the lung can not fill.

When there is present purulent or ichorous exudation in the thorax, the pleura will, if long exposed to its action, undergo necrosis, and a canal may be tunneled through the lung into a bronchus, and through this there may be more or less discharge, and a cure be ultimately effected. Caries of a rib may follow necrosis of a portion of the costal pleura, and a fistulous communication be opened up externally, the pus draining off, a cure being ultimately effected, or the prolonged suppuration may lead to tubercular deposit or to amyloid degeneration of the organs. A fatal peritonitis is in rare instances lighted up by the passage of ichorous matters through the agency of the lymphatics of the diaphragm. In other cases a fistulous communication is established, and the pus dissects downward along the psoas muscle, pointing under Poupart's ligament, or opens about the umbilicus, etc. Again, the pus may ulcerate into the mediastinum, into the pericardium, or into the great veins, but these are excessively rare accidents.

Chronic pleurisy differs only in time and extent from the acute form. In *pleuritis deformans* the exudations are of great thickness and extent, and, by adhesion and subsequent contraction, extensive deformity of the lung may result. The space left between the ribs and the lung will be filled with fluid, and, as the pleura is damaged so that absorption can not take place, encapsulation may hold the fluid months, even years. Often, indeed, the false membrane which has become organized possesses the power of pus-forming (pyogenic membrane), fistulous communications are established, and matter is discharged for years even. The chest becomes greatly deformed by shrinking, the shoulder depressed, the spine curved, and the heart pushed aside and permanently fixed in its new position.

Symptoms.—The symptomatology of pleurisy varies with the form. As *dry pleurisy* is the simplest form, it will be best to consider it first. This may set in with chilliness, fever, pain in the side, and dyspnœa, but more frequently there is little or no fever, no respiratory disturbance, only the pain in the side to indicate the nature of the attack. If the former symptoms are present, they do not continue longer than thirty-six to forty-eight hours; if the latter, the symptoms rarely necessitate confinement to bed. The physical signs of dry pleurisy are as follows: On inspection, the extent of the inspiratory movement is seen to be lessened by the pain—is arrested midway by a sudden start, and the body is curved a little to the affected side to avoid pressure on the inflamed membrane. On percussion, there is no change in the sonority from the normal minimum, because of the limited movement in inspiration, and if the pain is slight there will be no change in the normal maximum. On auscultation, the respiration will be feeble on the affected side, because of the pain elicited by the expansion in inspiration; and, if the pain is severe, the inspiratory murmur is rather suddenly arrested before completion, but if the pain is slight there will be no change in this respect. During the first two or three days, there will be audible on auscultation a sound due to the rubbing together of the roughened surfaces of the pleura—a *friction* or *to-and-fro rubbing sound*—synchronous with the respiratory movements, and ceasing when they are arrested. If strong and loud, this friction-sound produces a vibration of the chest-walls, or *fremitus*, which is recognizable on palpation. Dry pleurisy terminates in two ways—by resolution, or by adhesion. When resolution takes place, the pain and fever subside, and the friction murmur gradually lessens, and finally disappears. At the apex, the friction murmur modifies into a leather-creaking sound, persists, and may be confounded with the crackling *râles* which accompany the first stage of tubercular deposition—a mistake all the more likely, since pleuritic attacks are invited to the apex by the irritation of tubercle. Dry pleurisy occurs at the side and base of the thorax. This is the origin of the adhesions found after death, consisting of firm, strong bands of connective tissue, and which excited no symptoms that attracted attention. These bands often do serious mischief by limiting the movements of the lung.

Acute pleurisy with effusion, the ordinary form, sets in as any other acute inflammation, with chill, general *malaise*, and fever, with pain in the side; or there is in other cases, for several days, a daily paroxysm of fever, but without any local symptom for the first few days; or, again, there are cases in which pain in the side and effusion have preceded the febrile movement. Less often than pneumonia is pleurisy announced by a decided chill; more frequently there is chilliness recurring irregularly for the first few days. The fever which follows is a continued fever, with an evening exacerbation, and continues up to the beginning of the effusion, or about eight days, with little variation. If there are rigors occurring every day, although irregularly, and persist, it is probable that the effusion is purulent, or that the pleuritis is tubercular. The type of fever is not peculiar to the disease, and is not therefore diagnostic; the temperature does not often exceed 104° Fahr., and ranges from 101° to the former point. The pain is usually acute, lancinating, circumscribed, and is increased by breathing, coughing, or abrupt movements of the body. It is felt in the outer and inferior portion of the mammary region, sometimes at the base of the thorax, occasionally in the lumbar and iliac junction, and over a space which may be covered with a finger or two. It is commonly designated "a stitch in the side." Instead of being circumscribed, it may be diffused and ill-defined. The duration of the pain is variable; it may cease in three or four days; it may reappear after having ceased for a time; it may persist throughout the attack, and so long as it is present it affords evidence of the persistence of the inflammation. The severity and tenacity of the pain indicate the violence of the disorder. Dyspnœa is also a prominent symptom in pleuritis. Several factors are concerned. When the pain is severe, the inspiration is suppressed, shallow, and frequent; hæmatosis is accordingly impaired, and respiration is embarrassed from this cause. Fever, by increasing the waste of tissue and the excretion of carbonic acid, augments the necessity for oxygen. When effusion occurs, the respiratory field is narrowed, and mechanical difficulties are created by the pressure. The *decubitus* of the patient is highly characteristic. Before effusion has taken place, the position on the sound side is easier, for, as Traube has pointed out, the blood gravitates from the diseased side, and thus relieves the nerves of pressure ; but, when the effusion begins to compress the lung, the position on the diseased side becomes the easier. When there is extreme pressure, the patient can not lie down, and hence seeks rest in the semi-erect posture. More or less *cough* is present in pleuritis, and from the beginning. It is a suppressed cough, and is arrested in the act of inspiration by the catching pain in the side, and is again suddenly arrested in the explosion on account of the pain given by the shock. When effusion comes on, the cough declines, but when there is considerable effusion cough is induced by the attempt to take a full inspiration, or by change of position. The *expectoration* consists only of a little frothy mucus, unless bronchitis coexists, which is not unusual. As there are anorexia and more or less interference with digestion in all febrile diseases, the waste of tissue proceeds rapidly—on one side insufficient supply, on the other increased oxidation. Emaciation, loss of strength, with the accompanying depression of the nervous system, are prominent among the objective symptoms in pleuritis. The countenance has an expression of weariness, anxiety, and exhaustion, and may be pale or cyanosed. The cyanosis is present if there is much orthopnœa ; but there may be more or less pallor, possibly significant of hæmorrhagic pleuritis, especially if it occurred suddenly. The urine is scanty, high-colored, has high specific gravity, and deposits urates abundantly.

Although the rational symptoms of pleuritis are very significant, they are not so precise and definite as the physical signs. Having described the former, we will now take up the latter. On *inspection*, the movements of the affected side are seen to be restricted, to be suddenly arrested, and with an expression of pain. When effusion is present, an enlargement of the affected side is discerned ; the intercostal spaces are less concave, are elevated to a level of the ribs, even rise above them, and no movement takes place in respiration, while the healthy side is abnormally active. On *palpation*, the absence of vocal fremitus is a very important and significant symptom. The fremitus of the voice is lessened as the effusion rises, to be entirely absent when the chest is distended. On the sound side the vocal fremitus is exaggerated. When the effusion is large, on palpation there may be fluctuation detected in thin subjects ; by tapping one side smartly, a wave traverses the liquid and is felt on the opposite side. The character of the *percussion*-note is much affected by the quantity of liquid present. When there is a moderate amount of effusion, the tension of the lung is increased and consequently the note is high-pitched, rather hard, and having a distinct tympanitic quality. The tympanitic and high-pitch quality of the note is particularly evident on percussion of the infra-clavicular region, while the note becomes deeper and harder over the inferior and dependent parts where the effusion gravitates. So different are the pitch and quality of the percussion-note in the infra-clavicular region of the diseased and the healthy side, that, if the examination be carelessly made, the latter region, having none of the tympanitic quality, will appear to be diseased. When the fluid accumulates so that the lung is covered by a layer of fluid, two inches in depth, the percussion-note will be dull all over the chest, except at the sterno-clavicular articulation, where the note will still be high-pitched and tympanitic, although somewhat dull. There will be absolute dullness over the whole of the affected side, except posteriorly over the root of the lung, when the cavity is full and the lung flattened against the spinal column. Exception should also be made of a point corresponding to the junction of the second rib with the sternum, where a tympanitic note—*le bruit de pot fêlé*—indeed, is obtained by vibration of the column of air in the primary bronchus and trachea; but in both situations a high pitch and hard quality are the characteristics, if the lung, is entirely flat provided the percussion be lightly made, so as not to develop the tympanitic quality obtained from the trachea and bronchus. The value of the percussion-note is increased by the absence or presence of a *sense of resistance.* When there is fluid in the thorax, the sense of touch receives a different impression from that produced by the normal condition. The diagnosis of effusion in the left thoracic cavity is much facilitated by an attentive examination of the character of the dullness in the left hypochondrium. Owing to the shelving margin of the lung, but especially to the proximity of the stomach and large intestine, the inferior portion of the left lung returns a rather higher pitched and tympanitic note on percussion than the portion above. This space is about two to three inches in width at the lateral border of the chest, narrowing to nothing at either extremity. When fluid forms, the diaphragm descends by pressure, and this space is gradually encroached on, and in the case of large effusion disappears. In the first stage of pleuritis the respiration is jerking, and on the affected side the lung is imperfectly filled with air. On *auscultation* these

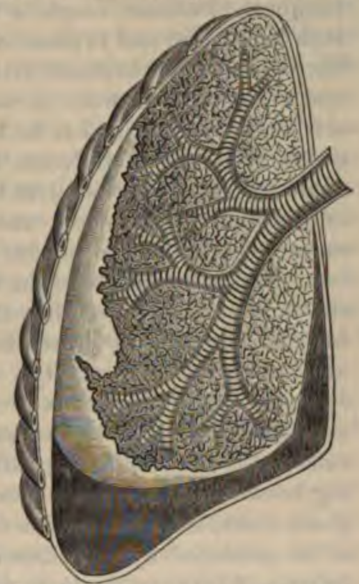

FIG. 27.—Limited Effusion and much Fibrinous Exudation. (Da Costa.)

characteristics of the breathing are ascertained — inspiration has a catching or jerking impulse, and hence the inspiratory vesicular murmur is feeble, because the lung can not be filled with air. When the membrane becomes rough, a rasping, grating murmur, audible with both inspiration and expiration—a to-and-fro friction murmur—is produced ; it is synchronous with the respiratory movements and ceases when they are arrested. It may be so loud and strong as to produce a friction fremitus, and to be heard away from the chest-wall. It becomes feeble as the effusion increases, and then disappears, to recur again for a short period after the fluid is absorbed. With the increase of the fluid in the chest, the vesicular murmur becomes more and more feeble and then ceases, and, when it is no longer audible at the base, may be heard above the line of effusion and of dullness. When the lung is compressed but the bronchi are still permeable, and the body of fluid not too great, the breathing has the bronchial character, and has no vesicular quality. When the lung is flattened against the spine, no breathing-sounds of any kind remain. Similarly, *bronchial voice*, or *bronchophony*, is audible from the still pervious bronchial tubes, as is the bronchial breathing, but this ceases as the corresponding breath-sound does, and no voice-sound remains. *Ægophony*, or goat's voice, is a modification of bronchial voice supposed at one time to be produced by the vibrations of a rather thin stratum of fluid, interposed between the chest-wall and the lung, but it is now regarded as a simple modification of broncophony. With the disappearance of the effusion the lung expands, and there is a gradual diminution of the dullness, until the percussion-note becomes normal and the resistance declines correspondingly. The vocal fremitus is restored in the same order. The voice and breath sounds are at first bronchial, then gradually become vesicular. As the bronchial voice and breath-sounds become audible, the friction-sound appears and continues up to the full restoration of the vesicular. Besides the friction to-and-fro sound, there are often heard, after the disappearance of the liquid effusion, coarse, creaking, grating sounds, which appear to be produced by the stretching of bands of adhesion, or the rubbing together of the large masses of solid exudation yet remaining for absorption. The author has witnessed the development and gradual disappearance of these sounds, during many months after recovery. Besides these sounds, *râles*, rather coarse, sub-mucous, and sub-crepitant, are audible during the process of absorption, and were supposed to be due to changes in the pulmonary parenchyma, but are now known to be produced by the opening up of tubes long compressed. Besides these, *râles* are present in cases of acute pleuritis, because of an accompanying bronchitis.

Course, Duration, and Termination.—Pleurisy does not pursue a defined course, nor does it terminate in crisis, which is the normal mode for pneumonia, but under favorable circumstances the development is gradual, and the return to health is by slow stages. Beginning in some one of the modes described, the fever regularly increases for the first four or five days, and then continues for eight or nine days pretty constantly at a uniform height. Then comes the period of effusion, when the temperature falls, the pain subsides, and the dyspnœa diminishes unless there is a large effusion, when the difficulty of breathing is proportional to the amount of compression to which the lung is subjected. The length of the time the effusion continues at its maximum varies from one day to five. The absorption may take place quite rapidly at first, but it does not continue at the same rate after the first two or three days. The reason is, probably, because the liquid part of the exudation is more easily disposed of, the solid portion needing to undergo a fatty transformation to fit it for absorption. The rate of absorption is measured by the gradual return of the normal sounds, by the diminution of the dullness, and by the movement of displaced organs to their proper positions. The changes in the condition of the inflamed parts are represented in the improved appearance, better appetite, and increasing strength. A marked change takes place in the urinary secretion, which becomes more abundant, less highly colored, and contains for a brief period cast-off epithelium and a trace of albumen. The absorption of the last part of the exudation is exceedingly slow, and months, even a year or two, may elapse before the physical signs indicate complete restoration. The return toward health is often interrupted by fresh attacks of inflammation, by a new outpouring of effusion, by an accession of fever and respiratory disturbance. Additional inflammation of the pleura and of the neo-membranes arrests the process of absorption, depresses the vital forces, and prepares the way to the chronic state, yet it sometimes happens that the new excitement awakens renewed activity in the process of absorption, which goes on more rapidly afterward. If, after the twenty-fifth to the thirtieth day, there is no appreciable diminution in the state of the effusion, the acute stage ends and the chronic begins. It may be that the effusion remains stationary, and the general condition continues good ; in other cases grave symptoms may arise, the temperature may increase, and in a day or two attain to the maximum of the first two weeks, or pass beyond it ; rigors may occur irregularly, followed by paroxysms of fever and sweats ; the countenance becomes anxious ; the tongue dry ; the depression great—without there being any change in the extent of the effusion or any new complication. This grave change in the condition of the patient is due to the purulent transformation of the exudation. It has already been indicated that the exudation may be purulent from the beginning, and that under these circumstances the symptoms have at the outset the septicæmic character above described. The termination is in *resolution ;* in the *chronic form ;* in *death.* The average duration of an acute, uncomplicated case is two to four weeks. Death may occur within the first two weeks, in the so-called fulminant form, or, when there is a very extensive sero-fibrinous effusion causing fatal syncope, most probably by compression of the great venous trunks, especially of the ascending vena cava, which may be twisted and its lumen obstructed by displacement of the heart. Again, œdema of the sound lung may suddenly ensue as a result of compression of its fellow, and cause death. An early recovery from pleuritis with effusion signifies that the effusion must have been of small extent. Any large inflammatory effusion, especially if the solid portion of it is considerable, must require a long time, months certainly, to dispose of it entirely.

Chronic pleurisy is an outcome of the acute disease, or it occurs primarily. It differs from the acute merely in the severity and chronicity of the symptoms. The fever is slight, the pain is not severe, but yet extensive changes will take place in the pleura. When the characteristic anatomical alterations have been effected, there will be fever of the septicæmic type. The rational and physical signs are the same as those of the acute form. The duration of the cases varies from two or three months to several years. Attempts at absorption going on favorably may be stopped by a new inflammation of the pleura, and of the neo-membranes with more effusion. An effusion that has remained stationary for a long time may, unexpectedly, undergo absorption by reason of the development of vessels in the new formations. But a cure by absorption is rare ; there are usually incomplete absorption, retraction and deformity of the chest, and permanent displacement of organs, or an external fistula, occurring spontaneously or resulting from an operation, may produce a favorable result comparatively. Without the operation of paracentesis, chronic pleurisy usually proves fatal by tuberculosis, by purulent infection, or by penetration of the pus into neighboring cavities, etc.

Complications.—The inflammation may extend by contiguity, and attack the pericardium—a not uncommon complication. There will occur a fibrino-serous exudation, often of considerable extent. The lung may be involved, but pneumonia is rather a coexisting disease—pleuro-pneumonia—than a complication. It is important to note that the lung on the sound side may be affected by œdema, a complication which adds immensely to the gravity of the case. Not only is the organ œdematous, but it usually presents patches of commencing pneumonic infiltration. The importance of pleuritis as a cause of phthisis is hardly sufficiently recognized, in inducing tubercular deposit, and by adhesions limiting the movements of the organ, thus inviting disease.

Diagnosis.—The most important difficulties in diagnosis are experienced in the differentiation of pleurisy with effusion from conditions in which the lung is solidified or is displaced by tumors, cysts, etc. Pleurisy is distinguished from *croupous pneumonia* by reference to the rational and physical signs. Pleurisy begins by chilliness, which persists for several days—pneumonia by a severe rigor, rarely two; the pain in pleurisy is a stitch, a lancinating pain, which can be covered by the finger—pneumonia by a sense of soreness and pain much more diffused; the fever in pleurisy is continuous—in pneumonia there is a distinct crisis or lysis, somewhere from the fifth to the eleventh day; the duration of pleurisy is indefinite—of pneumonia self-limited; the expectoration in pleurisy is simply frothy mucus—of pneumonia, rusty or bloody; in pleurisy the vocal fremitus is absent—in pneumonia it is not only present but exaggerated; in pleurisy there is a friction-sound, no crepitant *râle*, and the bronchophony is not so well defined—in pneumonia there is no friction-sound, the crepitant *râle* is present, and broncophony is loud and clear; in pleuritis there is more decided dullness, the intercostal spaces are pushed out, the thorax enlarged—in pneumonia the percussion-note is not so flat, the intercostal spaces and the size of the thorax remain normal; in pleuritis the organs are displaced; in pneumonia the relation of the organs is unaffected. Finally, the subsequent behavior of pneumonia and pleuritis leaves no room for doubt. An abscess of the liver pushing up the diaphragm, or an echinococcus-cyst growing in the same direction, of sufficient size to displace the lung in the same way, will cause the physical signs of an effusion into the thorax, and the diagnosis is possible only by a careful study of the history, which is entirely different in the two affections. A tumor or cyst of the chest will produce dullness on percussion, displace organs, and, by compressing the lungs, cause the disappearance of the voice and breath sounds. The differentiation is to be made by reference to the history of the cases, by the situation of the dullness toward or about the central and superior parts of the chest in tumor—the inferior part of the chest in effusion; by the general and symmetrical bulging of the chest-walls in effusion, the circumscribed and irregular bulging caused by tumor; by the absence of vocal fremitus in pleuritis—its exaggeration in cases of tumor.

Although the withdrawal of the fluid is the only certain means of arriving at the nature of the effusion, there are signs by which we may approximate with considerable accuracy to a correct diagnosis. If, during the acute stage, the fever running high, the effusion pouring out rapidly, there suddenly ensue great pallor, weakness, and depressed temperature, followed after some hours by rise of temperature even higher than before, a hæmorrhage has probably occurred; or, if during the chronic stage there are recurrent attacks, and the above-described symptoms occur, the case is not only hæmorrhagic, but the underlying morbid process is tuberculosis. If the case is characterized from the beginning by repeated rigors, occurring irregularly, and followed by paroxysms of intense fever and sweats, the exudation is purulent; if, during the course of an ordinary attack of sero-fibrinous pleuritis, the same septicæmic symptoms arise, the exudation has been transformed into the purulent.

Treatment.—As soon as the pleuritic inflammation begins, and the pain is a good indication, the patient should receive a full dose of quinine and morphine (℈j quinine and gr. ss. morphine for an adult), and the effect of this should be maintained by the repetition of smaller doses (gr. v quinine, $\frac{1}{8}$ gr. morphine) every four hours. If the stomach is irritable, the morphine can be administered subcutaneously, or, if the pain is very acute, this mode of administration is more effective than by the mouth. Besides the power of morphine to relieve pain, it is an effective remedy in serous inflammation. The combination which was so much employed formerly (calomel and opium) owed its virtues to the opium. If constipation is a feature of the case, especially if nausea accompanies it, and if there be evidence of congestion in the portal circulation, the most important results may follow the exhibition of a sufficient dose of calomel. The dose should be, as my experience goes, from three to five grains, and the reasons for its use, are that calomel has a distinctly sedative effect on the liver, lessens the physiological activity of its constructive apparatus, lowers the temperature of the blood passing through the hepatic veins into the general circulation, and it depletes, by its purgative action, the portal system. In consequence of these effects the whole system experiences a sense of relief, and the local morbid process is much ameliorated. That calomel has germicide powers, of itself, or by the conversion of some part of it into the corrosive chloride, we may accept, also, as a part of its singular efficiency under the conditions mentioned. If there be much fever—a strong pulse and elevated temperature—the stomach not irritable, and the subject be plethoric, a dozen cups or leeches, drawing six ounces of blood, can be applied with advantage. The old plan of bleeding *ad deliquum animi*, or until the pain ceased, was a powerful and certain means of relieving pain which has been rightly abandoned, but the local bloodletting is of service and not unfrequently general venesection is useful. The blood-pressure can be reduced also by active purgatives, of which the salines are best. When the exudation is poured out, a different plan will be necessary. The only agents which possess the property of dissolving an exudation are the alkalies, and the most efficient of these is ammonia. Carbonate of ammonia can be best given in a solution of the acetate (gr. v—x in ℥ ss.—℥ j). They should take the place of the quinine and morphine. Absorption will be much aided by keeping up free outward osmosis through the intestinal mucous membrane by saline laxatives. The same process can be carried on through the skin by the use of pilocarpine. This should be administered, as a rule, once a day, but its action on the heart should not be forgotten, and care exercised if there be displacement of this organ, especially if there be a twist in the vena cava. The best mode of administering pilocarpine is the hypodermatic injection, pilocarpine—$\frac{1}{8}$ of a grain of any of the salts. As the pouring out of so much fluid, the waste of tissue produced by a high temperature, and the interference with assimilation caused by the disordered digestion, rapidly impair the vital forces, it is important, by proper food-supply and the judicious use of stimulants, to obviate the asthenia. When, however, a large effusion exists, especially if purulent, it becomes necessary to remove it by the operation of thoracentesis. Even if absorption may eventually succeed in disposing of the fluid, there is great danger that the lung will not be in a condition to expand again fully, and retraction and deformity of the chest will be the result. If the effusion be purulent, absorption can not take place, and hence thoracentesis is indispensable. The question of how early shall thoracentesis be performed has been much discussed. It ought not to be undertaken within a few days after effusion, nor unless the symptoms of compression are urgent while the exudation is going on. It ought not to be performed if the natural powers are equal to the task of removing the fluid early enough to save damage to the organs concerned. These rules apply to the sero-fibrinous form of pleuritis. Thoracentesis ought to be performed in the purulent form as soon as the nature of the case is evident, for nothing is to be gained by delay. The point of election when the choice may be made is underneath the inferior angle of the scapula, but the needle may be inserted at any place with due regard to the position of the heart and great vessels. As regards the method of procedure, nothing has been added practically to the method of Bowditch (the real inventor of the *aspirateur*), which consists in exhausting the chest by the pump and attached needle. Although the admission of air does not seem to be very important, yet it is better to avoid it in cases of the sero-fibrinous, for, if subsequent operations are necessary, the effusion will become more and more purulent. If this is the case, the tincture of iodine or a diluted compound solution can be injected with great advantage after removing the fluid (liq. iodinii comp. ℥ j—aquæ ℥ iv). This iodine injection is highly useful in empyema.* Precautions to avoid air are usually regarded as unnecessary in the case of purulent effusion. In those cases requiring repeated tapping, late experience has shown that the best results are obtained by establishing free drainage. If a sufficient opening for the drainage-tube can not be obtained in the intercostal space, exsection of the rib is then necessary. The simplest of these operations should be performed with antiseptic precautions. If the pus of an empyema undergo decomposition and become foul, the cavity should be freely washed out with antiseptic precautions. Although the admission of air in cases of empyema is not sought to be prevented, nevertheless the air should be deprived of its germs of putrefaction.

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* A warm solution of chlorate of potassa (ʒ j or ʒ ij—O j) or of salicylic acid and borax (ʒ j of each to the O j), may also be used to wash out the cavity in empyema.

As death has occurred several times very unexpectedly after the operation of thoracentesis, certain precautions are necessary. When the effusion is large, the whole amount should not be withdrawn at once, for the sudden removal of the pressure might induce a quick outpouring of fluid, or the great vessels, relieved of pressure, would overdistend the right cavities, or the heart, moving from its position, might cause compression of some of the vessels. Sudden death might very unexpectedly be caused by any of these accidents, notwithstanding the operation of thoracentesis is simple, not painful, and is otherwise free from danger. After the removal of the liquid exudation by absorption or by thoracentesis, a quantity of solid and semi-solid remains behind and is very slowly transformed. A succession of flying-blisters, painting with the tincture of iodine, and friction of the affected side with ointment of the red iodide of mercury, are the most effective external or topical applications. The best results are obtained, not from the use of supposed stimulants of the absorbents, but from means to promote the nutrition. The iodide of iron (sirup), cod-liver oil, extract of malt, and a generous diet, the digestion stimulated by bitters and mineral acids, are the best means for increasing absorption. The amount of fluid taken should be reduced to the minimum ; for, although the restrictions imposed in a "dry diet" may be too rigid for ordinary patients, yet they can submit to a considerable reduction of the fluid. Absorption is promoted by lessening the water of the blood, which can be accomplished by saline laxatives and jaborandi. The laxatives should not be given so as to interfere with digestion, and a daily dose of jaborandi can be so administered as not to interfere with the appetite or exercise. To procure complete distention of the lung, and to promote the oxygenation of the blood, compressed air should be inhaled daily, or a sojourn in an elevated, dry mountain-region should be enjoined. Although we may not agree with Dr. Leaming, of New York, and Dr. Andrew Clark, of London, in the importance of pleuritic exudations as a factor in phthisis, we must admit that they exercise some influence in initiating the process of fibroid degeneration and of tuberculosis.

HYDROTHORAX—DROPSY OF THE CHEST.

Definition.—By the term *hydrothorax* is intended an accumulation of watery fluid in the chest. It differs from pleuritis in the character of the fluid and in the state of the pleura. In pleuritis the effusion is an inflammatory exudation, and the pleura is the seat of an inflammation ; in hydrothorax the fluid transudes—a merely physical process—and the pleura is unaffected except by maceration.

Causes.—The various conditions giving rise to general dropsy will cause hydrothorax—cardiac and renal diseases. Local obstruction to the course of the circulation produces pure hydrothorax, i. e., hydrothorax not a part of a general dropsy. The most important of these local causes are emphysema and sclerosis of the lung, tumors so situated as to compress the vena cava, vena azygos, the right auricle, etc. A general dyscrasia may induce hydrothorax, as Bright's disease, chronic malarial poisoning, etc. The most influential factor is the condition entitled by the older authors *latent pleurisy*. In this malady there is a state of the pleural membrane closely allied to pleuritis—to that form known as *dry pleurisy ;* but instead of a plastic exudation there is an abundant outpouring of serum.

Pathological Anatomy.—When the hydrothorax is due to any of the causes producing general dropsy, the effusion is bilateral, but usually more abundant on one side. There will be found associated with the hydrothorax the anatomical changes in the lungs, heart, and kidneys, proper to the particular form of dropsy. The fluid has a pale sea-green color, is transparent, and frequently coagulates on exposure to air, the coagulation consisting in the formation of an excessively fine reticulation of the minutest fibers. In the case of the so-called latent pleurisy the membrane is thickened, congested, and coated usually with a pellicular exudation, portions of which are, to a greater or less extent, floating in the fluid. The amount of serum present is from half a pint to two or three gallons. The effect of the fluid on the position of the heart and other organs is precisely the same as in pleuritis. The retraction of the lung and its subsequent compression also take place, as in pleurisy, except that it occurs more regularly.

Symptoms.—In latent pleurisy, so called, there is some pain felt in various parts of the chest, but it is not acute and well defined as in pleurisy. It is usually situated in the side, and is a rather dull, tensive, heavy pain, or a feeling of soreness. It is increased by a full inspiration, or by coughing, but is not so severe as to interfere with daily duties ; and it is often transient, and makes so little impression on the mind as to be forgotten until attention is directed to it. There is some feverishness toward evening, but not much attention is paid to it, and hence it is usually overlooked. The cough may be rather troublesome, especially on lying down, but the expectoration is nothing more than frothy mucus. Often these symptoms pass unnoticed, and the first thing which attracts attention is an increasing difficulty of breathing. In the cases of hydrothorax pure, without pleural inflammation, there is no fever, nor pain in the side, and the first symptom referable to the thorax is difficulty of breathing greater than in pleurisy, because the effusion is on both sides. In latent pleurisy, the left side of the thorax is involved in two thirds of the cases ; consequently the heart is pushed over to the right, and the semilunar space is obliterated. In hydrothorax there is no displacement of the organs, because of the effusion on two sides and in the abdominal cavity. The physical signs are much the same in hydrothorax as in pleurisy; but in the former there can not be that complete filling of the cavities, and hence there must be a considerable space of both lungs where the voice and breath sounds remain unaffected. Furthermore, in hydrothorax, there being no limitation of the effusion by neo-membrane and by adhesions, the fluid gravitates with the changes of position, and the area of dullness shifts accordingly. The course, duration, and termination of hydrothorax are those of the disease on which it depends. The formation of a large effusion in the chest adds to the severity of the case, and is not unfrequently a cause of death. This is especially true of dropsy, whether cardiac or renal. The hydrothorax is a source of extreme distress when it may not prove fatal, for the patient is unable to lie down, or to make any muscular effort without experiencing a suffocative attack. The author has witnessed a case of sudden death from hydrothorax in an aneurism of the arch of the aorta which was solidifying. The behavior of latent pleurisy is that of the sero-fibrinous form of acute pleurisy, when sufficient fluid has accumulated to produce symptoms by compression.

Treatment.—If there is large effusion, delay is unsafe and thoracentesis should be promptly performed. As serum will flow through a fine capillary needle, but little pain and no danger attend the operation of aspiration. If the effusion is not sufficient to produce distress by pressure, the treatment is directed to the condition on which the dropsy depends. The treatment for latent pleurisy is the same as for acute pleurisy with effusion. As the inflammatory symptoms are usually overlooked, the physician is not consulted until the difficulty of breathing comes on, and then the sole question is, aspiration or not. The rules for guidance are the same as those already laid down.

PNEUMOTHORAX—HYDROPNEUMOTHORAX.

Definition.—The presence of air in the cavity of the thorax is called *pneumothorax;* of air and fluid, *hydropneumothorax.*

Causes.—Air or gas of any kind is rarely present in the cavity without liquid, and if air alone should enter an exudation would soon be excited. It is now settled that a serous membrane can not secrete air, and that, therefore, if air be found in the cavity of the pleura, it came there from without, or is a gas the product of decomposition or fermentation. Almost always it enters from without by perforation of the pleura, by the lung, or by the wall of the thorax. The most frequent mode of entrance of air is the giving way of a superficial cavity of the lung, tubercular or caseous. Very rarely the air passes through a communication made by a gangrene patch, or a hæmorrhagic infarction, and still more rarely by the giving way of emphysematous alveoli. Abscesses of the liver ulcerating through the diaphragm may form a secondary purulent collection in the pleural cavity, which may communicate through the lung with a bronchus, constituting pyopneumothorax. One of the modes of termination of a purulent pleuritis is by a fistulous passage to a bronchus, through which air is admitted to the pleura. Suppuration may occur in neighboring organs in a way to involve the pleura and thus secure some outlet. For example : suppuration of bronchial glands, bursting into the pleura and ulcerating into a bronchus ; abscesses of the liver or of the kidney, perforating the diaphragm and the lung, etc. Traumatism is an important factor, pyopneumothorax being caused by penetrating wounds, incised or gunshot, the air entering from without.

Pathological Anatomy.—The accumulation of air in a given case is much influenced by the formation of the orifice of communication. If the entrance is easy and the exit difficult, a very large amount of air may accumulate, and very often a sort of valvular arrangement, a fibrinous flap or plug, may exist at the orifice which has this effect. The lung quickly retracts until there is an equilibrium of the pressure; compression is then exerted on it if the orifice is such that the air which entered without obstruction can not escape. The quantity of air which can be contained in the cavity depends on several conditions; on the compressibility of the lung, which may be slight, owing to solidification by caseous or tubercular deposits; the degree in which the other organs can be shoved aside ; the amount of liquid present, etc. It is a mixture of gases, not air, usually found in the cavity—of nitrogen and carbonic acid, and but little oxygen, with some sulphuretted hydrogen if there be unhealthy pus present. If atmospheric air enters, the pleura inflames, and sero-purulent, then purulent exudation is poured out. As air contains the bacteria of decomposition, they are the active agents in exciting purulent inflammation ; but, as, in pneumohydrothorax, ichorous, ulcerating, or decomposing materials pass in under the usual circumstances, these play a considerable part in exciting inflammation, though probably less than the air and its contained germs. The exudation which results from the action of these noxious matters is purulent, often ichorous and bloody. The gas is contained in the space above the liquid, and the lung, having had the air squeezed out of it, lies flattened against the spine, unless old and firm adhesions resist the compressing forces. If there be much fluid, that side of the thorax will be enlarged, the intercostal spaces prominent, the diaphragm depressed, the heart pushed aside, etc. In some rare instances adhesions form in a circle between the two pleural surfaces, making a central cavity in which gas and fluid will accumulate to a large extent, a fistulous communication having been established with a bronchus.

Symptoms.—Pneumothorax is to be studied in connection with the diseases from which it arises. It may develop insidiously, so that it is discovered only on making physical examination of the chest. But, when a perforation occurs suddenly, pronounced, even formidable, symptoms are at once produced. Perforation may be announced by a condition almost of collapse, a temperature of 97° Fahr., and a small, weak, but very rapid pulse. If the temperature does not descend so low, the pulse is weak and rapid, and the respirations are hurried—the former reaching so high as 140, the latter up to 40, even 60. At the same time dyspnœa sets in with orthopnœa, and a severe pain, due either to sudden stretching of the pleura or tearing apart of adhesions. In other cases, for example phthisical subjects, none of these severe symptoms are produced, probably because narrowing of the respiratory field has been going on so long as to prepare them for this additional discomfort. The decubitus varies, the largest number seeking a position on the diseased side to permit the freest possible play of the healthy lung; but a considerable proportion lie upon either side, although, when air first entered the cavity, orthopnœa was experienced by most of the cases. The dyspnœa is due to several causes—to sudden compression of the lungs and the heart, and to a compensatory congestion, often with œdema of the other lung, whence the expiratory force is lessened and the voice weak and trembling. Cyanosis appears if

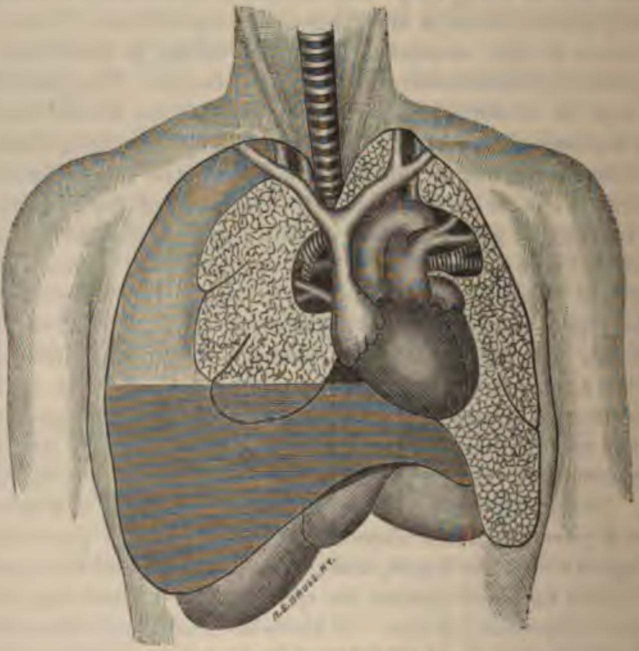

FIG. 28.—Hydropneumothorax.

there is much difficulty of breathing, the surface becomes cold and covered with a cold sweat, the tongue is blue and cold, and death soon closes the scene ; or, if life continues, general œdema supervenes from the venous stasis, while the arterial tension is low from ischæmia of the arteries. The lessening of the expiratory force makes the cough weak and ineffectual, and the expectoration diminishes. The low state of the arterial tension affects the urinary secretion, which is dense and red, with traces of albumen. The vocal fremitus may be present, diminished, or absent, in pneumothorax—present when there are strong bands of adhesion which communicate the vibrations to the chest-walls ; diminished when the lung is not entirely collapsed ; absent when the cavity is distended with gas. On palpation, also, increased resistance will be noted while there is fluid, and increased tension with diminished resistance where there is gas. The percussion-note is characterized by its marked tympanitic quality, resonance, and elasticity. The resonance is not limited to the part containing air, but extends downward to the lower margin of the ribs, extinguishing the hepatic dullness in its usual limits, and the semi-lunar space on the left side, and also extends across to the middle of the sternum. A peculiar metallic echo may be developed on strong percussion. Percussion over the fluid produces the usual dull sound which sharply contrasts with the metallic clang of the percussion over air, and the dullness here varies with the position of the patient and follows the gravitation of the liquid. The character of the percussion-note is affected by several circumstances : when thick, false membrane lines the thoracic wall it acts as a damper, and there is much less of the tympanitic and metallic quality ; when an external opening exists, there will be produced the cracked-pot sound. On auscultation, there is no respiratory sound, except a modified, amphoric, blowing sound. All of the sounds audible in the chest—cough, *râles*, heart-beat, etc.—take on a distinct metallic quality. The dropping of fluid, or coughing, or movements of the body, produce under these circumstances *metallic tinkling*. But the most characteristic of the physical signs is *succussion*—a splashing of the liquid against the walls of the chest, produced by a sudden shake of the body. It is best heard by applying the ear to the chest, and then suddenly shaking the body by the hand placed on the patient's shoulder. The patient often recognizes this sound, and soon learns the best movement to produce it. It is like the splashing of liquid in a half-empty barrel.

Course, Duration, and Termination.—The course of pneumothorax is much influenced by the associated lesions and the extent of the pulmonary insufficiency. If, already, the respiratory field is much narrowed, death may ensue in a few hours or days. Death is more frequently produced by the secondary pleuritis and its products, causing slow failure of respiration after some weeks. A cure is not to be expected in cases, the most numerous, due to perforation of a superficially placed cavity. Pneumothorax resulting from an incised wound in a healthy subject may get well after some weeks. A perforation occurring in the first stage of phthisis is not so important as one occurring later, and a cure is possible in the former before the constitutional forces are much depressed by the progress of the phthisis. A pneumothorax, produced by the discharge of a purulent pleuritis by a bronchus may get well after some months. It may be stated in general that the prognosis of pneumothorax is unfavorable, since very few cases get well even in the modified way of a permanent fistula.

Diagnosis.—Pneumohydrothorax may be confounded with the large caverns of phthisis, with dilated bronchi, with emphysema, with pleuritis having limited effusion. Vomicæ are confined to the upper part of the lung, have formed slowly without any sudden symptoms; they present amphoric sounds and metallic tinkling, rarely succussion; vocal fremitus is not lessened; the chest-walls are retracted instead of distended, and the heart is not displaced. In pneumohydrothorax, loud, deep, tympanitic percussion-note is obtained all over the affected side; the symptoms have occurred suddenly, and consist of severe pain, dyspnœa, and orthopnœa; well-marked succussion; vocal fremitus lessened or absent; the intercostal spaces bulging instead of retracted; heart and other organs displaced. Emphysema is bilateral; the respiratory murmur not absent; bronchial *râles* audible all over the chest; vocal fremitus present. Pneumohydrothorax is unilateral; the respiratory murmur entirely absent, and all voice and breath sounds and *râles* from the affected side wanting when the lung is collapsed; vocal fremitus absent. In pleuritis, with effusion, the percussion-note has a tympanitic quality in the infra-clavicular region; the dullness on percussion changes with the positions of the patient, and corresponds to the height of the liquid; an amphoric murmur is exceptionally audible over the root of the lung and at the summit; with the increase of the distention of the chest, there is absolute dullness over the whole side; no metallic tinkling, no succussion. In pneumohydrothorax, the percussion-note has a loud, ringing, tympanitic quality all over the chest, instead of a modified normal at the infra-clavicular region, and this tympanitic note is not supplanted by absolute dullness; there are metallic tinkling and succussion in perfection.

Treatment.—As respects the condition associated with pneumothorax and pneumohydrothorax, the treatment is indicated under the head of these maladies, and need not now be discussed. If there are much dyspnœa and danger of acute asphyxia, no time should be lost in making a free opening to permit the exit of air. The pyopneumothorax is to be treated by incision and the drainage-tube, and the use of antiseptic injections, of which iodine appears to the author to be the best. The severe pain requires the use of anodynes, unless the free exit of air procured by incision relieves the distress. The congestion and œdema of the sound lung may be relieved by ligatures to the thighs, by which a considerable quantity of venous blood can be retained in the lower limbs long enough to bridge over the period of danger. This expedient is preferable to bloodletting, which has been recommended for this purpose.

PNEUMONIA—PNEUMONITIS—INFLAMMATION OF THE LUNG.

Definition.—*Pneumonia*, an acute inflammation involving the alveoli of the lungs, is designated by the German writers "croupous pneumonia," and by the French writers "fibrinous pneumonia." "Catarrhal pneumonia" differs from the fibrinous or croupous form in the seat and character of the inflammation. It attacks the capillary tubes immediately next the alveoli, and is a catarrhal instead of a croupous inflammation. The so-called lobular pneumonia is nothing more than catarrhal pneumonia, the changes in the lobules being secondary to the catarrhal process in the ultimate bronchi. Lobar pneumonia is a fibrinous or croupous pneumonia occupying and confined to a lobe. Pneumonia is also known in common language as "lung-fever," "winter-fever," etc.

Causes.—There is a growing belief that pneumonia is a constitutional or an infectious disease, like typhoid or relapsing fever. It differs from other inflammations in that it is self-limited, and terminates by crisis. It is a very common disease; it occurs in all degrees of latitude, under every variety of climate, and at all ages. It is common in infants at the breast, but declines somewhat after the second year until after the second dentition, and is frequently encountered and is very fatal in the old. The male sex is most frequently attacked, because men are more exposed than women to those external conditions which tend to produce it. In-door life, a vitiated atmosphere, excesses, especially alcoholic, and bad hygienic influences of every kind which induce debility, favor attacks of pneumonia. Certain seasons appear to invite the disease—those parts of the year characterized by humidity, high winds, and low temperature. In the British Islands winter is the season of greatest prevalence; on the Continent, spring; in this country, winter and spring, the former especially—hence the name winter-fever. Occasionally, pneumonia occurs in so many persons in a particular district that it may seem to be epidemic, but there are atmospheric influences at work to produce the disease, especially excessive moisture conjoined with low temperature. It is a common belief that pneumonia is caused by exposure to cold, especially to draughts when the body is warm and perspiring. That catarrhal pneumonia is induced in that way no one will dispute, but it is more than doubtful that croupous pneumonia is thus caused, unless there exist a predisposition to it, either of a vulnerable constitution or an inherited tendency to pulmonary disease. A phthisical tendency, the author believes, is the chief factor, or that peculiarity in the structure of the pulmonary tissue associated with consumption. There are other diathetic states concerned in the production of pneumonia—as gout, rheumatism, diabetes, the eruptive fevers, especially chronic alcoholism. Probably the real etiological factor is a *microbe*. Klebs* first made the attempt to define the pneumonococcus, but the *micrococcus* pneumococcus of Friedländer has been more generally accepted as the true infective organism of croupous pneumonia, and this is active under the atmospherical conditions above mentioned, but the *dipplococcus* of Fränkel is held by some to be, also, an efficient agent in setting up of the pneumonic process.

Pathological Anatomy.—The state of the affected lung in pneumonia is usually divided into three stages, following the original description of Laennec, based on the naked-eye appearances: engorgement; red hepatization; gray hepatization. The better arrangement, based on the description of Jaccoud,† but modified, is as follows: *The stage of hyperæmia*, or engorgement; the *stage of exudation* (red hepatization); the *stage of resolution* (degeneration and extrusion of the exudation); the *stage of purulent transformation* (gray hepatization). In the stage of hyperæmia or engorgement, as now described, there are two distinct and separate acts—the increased blood-supply and the pouring out of an exudation. The lung has a reddish-brown appearance, is heavier, floats in water, but sinks lower than the normal lung-tissue, crepitates but little when pressed, and it is no longer elastic, but when an impression is made by the fingers it is retained. On section it presents a pretty uniform brownish-red tint, and it exudes a quantity of blood. On microscopic examination the blood-vessels are found to be distended with blood, and the capillary network surrounding the alveoli is so much enlarged that the alveoli are encroached on by it.‡ The adjacent portions of the bronchioles are similarly engorged, the mucous membrane dark reddish from fullness of the vessels. This hyperæmia marks the first stage in the inflammatory process. The next step consists in the *pouring out and coagulation of an exudation*. There is exuded into the alveoli an albuminous or fibrinous fluid of great viscidity, and with it leucocytes which have wandered from the vessels, and red blood-globules present by diapedesis, and blood by the rupture of distended capillaries. This viscid albuminous fluid is poured out also into the bronchioles and bronchi of the inflamed section, and with it leucocytes and some red corpuscles. When the surfaces approximate, this adhesive fluid holds them tightly together until the incoming air separates them. In the capillaries of the inflamed area the blood-current is finally stopped, and the corpuscles are then seen to be closely packed together and flattened at the points of contact. The albuminous or fluid exudation remains fluid for a short time, and then solidifies or coagulates, beginning in the alveoli and extending through the bronchioles outwardly. In coagulating it incloses the white and red corpuscles, and fills out the alveolus or bronchiole, probably expanding somewhat in the act of coagulation. When this process is completed, the inflamed part is solid, entirely without air, and falls immediately to the bottom when placed in a vessel of water; it is also friable, is easily broken up between the fingers, and on section with the knife divides cleanly with well-defined margins. The cut surface presents a reddish color, and is granulated; this granular appearance being due to the little masses of coagulated exudation filling the cavity of the alveoli. These little masses may with some care be lifted out of the mold in which they are formed and held on the point of a pin. The tissue of the inflamed part, in respect to color, density, and granular appearance, so strongly resembles the cut surface of a section of the liver as to be called by Laennec *red hepatization.*

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* "Archiv für experiment. Pathologie u. Pharmacol.," vol. iv, p. 1,1875.

† "Traité de Pathologie Interne," vol. ii, p. 45.

‡ Rindfleisch, *op. cit.*

There are two directions which the inflammatory process may now assume: toward resolution, or return to the normal state; toward purulent transformation. As the first is the more usual, we describe first the *process of resolution.* The albuminous material which had solidified undergoes liquefaction, and the pressure is thus removed from the surrounding vessels. The watery parts of the exudation diffuse into the vessels, and the solids, together with the cellular elements, undergo a fatty metamorphosis, and are transformed into an emulsioned mixture without any of the viscidity of the original exudation, and capable either of absorption or of extrusion, much of it, doubtless, being expectorated. As the exudation liquefies, air again enters the alveoli, diffusion of oxygen into and of carbonic acid out of the blood is resumed, and the current of the circulation is fully reëstablished. The effusion into the connective tissue between the alveoli and bronchioles is finally taken up, and the normal color and density are restored to the inflamed part, but its elasticity continues impaired for a long time.

When the *purulent transformation* takes place, a change is wrought in the density, color, and constitution of the inflamed area. It has been much discussed whether the epithelium of the alveoli undergoes any change, and contributes, by multiplication of its cells, to the exudation in croupous pneumonia, and whether any of the pus-corpuscles which become so abundant during the stage of gray hepatization or purulent transformation originate by proliferation of the epithelial cells. The former is denied by most authorities; the latter is highly probable; but the pus-cells are derived chiefly from the wandering white cells by multiplication and division. With the formation of pus-cells a process of fatty degeneration takes place in the albuminous exudation, but the rapid and exuberant formation of pus-cells is the principal event, the tissue being changed in color from the reddish-brown appearance of the red hepatization to the yellowish or grayish-yellow tint of gray hepatization. When such tissue is squeezed a little, a quantity of pus exudes, and the whole is easily broken up into a fatty and granular mass. Not all parts of the inflamed area are equally advanced in suppuration, some parts still preserving the reddish-brown, with here and there a patch of yellow; and others uniformly grayish-yellow, and some still advanced beyond this into a yellowish, almost diffluent mass. The stroma of the lungs yet remains intact, notwithstanding the enormous production of pus-cells. In rare cases a portion of the affected tissue proceeds beyond the stage of gray hepatization, or purulent transformation; the stroma of the lungs yields, becomes disintegrated, and a small purulent collection is formed. A large abscess may be formed by the coalescence of several smaller ones. The collection may be bounded only by disintegrating lung-tissue, or the pus may be inclosed by a limiting membrane, or, in other words, become encysted. The author has seen a case of encysted abscess occupying a part of the middle of the right lung, which had existed for several months without symptoms. They may discharge by a bronchus, or into the pleura, or the pus of the encysted abscess may gradually undergo absorption. The termination by gangrene is much more uncommon than that by abscess, and, when it does occur, signifies a most depraved state of the tissues. The passage of acute into chronic pneumonia is a comparatively frequent occurrence, when the disease is of diathetic origin, especially in strumous subjects, or when a tendency to pulmonary disease exists. When the change to the chronic form takes place, the process of retrograde metamorphosis of the exudation preparatory to its extrusion is arrested; the tissue appears compact, grayish, with here and there dark patches of pigment; the hyperæmia has ceased, and the infiltrated liquid is absorbed. In other cases the whole of the inflamed area does not pass over to the chronic stage; resolution takes place more or less perfectly; the exudation is disposed of in part, but still portions remain, more or less impairing the functions of the part. In other cases the products of inflammation are transformed into caseous matter. This change occurs when purulent transformation has taken place. The pus loses the fluid in which the corpuscles float, and these bodies become fatty, and more or less calcareous matter is mixed up with the fat, the ultimate product being a soft solid, looking like and having the consistence of cheese—whence the term *caseous matter.* It must be stated that this termination to croupous pneumonia is regarded by the best modern authorities as very uncommon, while it is usual to catarrhal pneumonia. All parts of the lung are not equally susceptible to the pneumonic inflammation. The statistics show that the right lung is affected alone in one half of the cases, and as regards the left nearly twice as often, or, to express the relation more definitely, using the statistics of Juergensen—the right lung was affected in 53·7 per cent., the left lung in 38·23 per cent., both lungs in 8·07 per cent. The inferior lobe of the right lung is the point of election, being the seat of inflammation in three fourths of the cases. There are certain consequences which follow on a pneumonia that ought not to be overlooked. When a considerable part of a lung suddenly ceases to functionate, there must be disturbances set up in its fellow. The obstruction to the pulmonary circulation induces over-distention of the right cavities and the veins, and ischæmia of the arteries. The blood displaced from the inflamed part, and which can not circulate through it, induces hyperæmia and œdema of the other lung.

Symptoms.—There are two modes of onset: in the less frequent there has been a day or two of bronchial catarrh and general *malaise*, when some chilliness is experienced, pain is felt in the side, and the disease proceeds in its usual way. In the other and more frequent mode, a decided rigor is the initial symptom—a rigor more severe than in any diseases except malarial fever and pyæmia. Elevation of temperature occurs at once, and by the evening of the first day has reached about 104° Fahr. In infants, instead of chill there may be a violent general convulsion or several of them. The duration of the cold stage is from a quarter of an hour to three or four hours, and during it the thermometer in the axilla notes some slight elevation of temperature, and in a few hours not only is the external temperature high, but the subjective sense of heat is great. The face is flushed, the eyes injected, there are intense headache, severe pains in the back, and muscular soreness in the members. The pulse is large in volume and strong in tension. There is usually a whitish-coated tongue, the appetite is wanting, and the stomach is nauseated, or there are attacks of vomiting on the first day. By the end of the first day, or the beginning of the second, there are rational symptoms which indicate the chest as the seat of the mischief. Pain in the side is experienced, and difficulty of breathing and cough now come on. The pain in the side varies in severity, and indeed is not always present. If the pleura is involved, the pain is more prompt and more acute ; if the deepest part of the lung, there may be no pain until the inflammation approaches the surface. The pain is most severe when it is first felt, and then it usually declines. The position of the pain is, as a rule, in the right chest, a little below and external to the nipple, but it may be felt in the lumbar region, in the iliac region, and in the shoulder. When pneumonia has attacked the summit of the lung, or as it occurs in the aged, pain may be absent. Coughing, breathing, especially a deep expiration, increase the pain. Accompanying the pain, or coming soon after it, is dyspnœa ; the respiratory acts are more frequent and shallow, reaching as high as thirty or forty per minute, the shallowness being due to the pain caused by full breathing, and by the narrowing of the respiratory field. The flushed, anxious, and somewhat dusky countenance, the working of muscles of respiration merely accessory, and those of the alæ of the nose, make up an expression which has been called *facies pneumonica.* The cough, which appears on the first or second day, is very characteristic ; it is husky, suppressed, and painful. At first there is brought up a little frothy mucus, but on the third day there appear the sputa characteristic of this disease ; thick, viscid material like that which is poured out and coagulates in the alveoli and bronchioles of the lung. The sputum also contains blood-corpuscles intimately incorporated with the viscid albuminous matter, but in varying proportion of coloring, from a light brick-red to a brownish-black. So tenacious and adhesive is the sputum that it remains adherent to the bottom of the vessel if turned over, and if a considerable quantity is collected in a vessel it presents a jelly-like appearance of consistency. The blood is not always mixed with the sputa at first, but the peculiar characteristics of the expectoration are in other respects present, the blood appearing in four or five days. In some debilitated subjects—for example, the subjects of chronic alcoholism—the expectoration is thinner and more abundant, presenting an appearance like prune-juice, whence the name *prune-juice expectoration*—an ill-omen. Again, there may be no expectoration at all, which is sometimes the case in very adynamic states, and in pneumonia of the apex. There are also present in the sputa casts of the finer bronchi. The sputa should be agitated with water, and the grayish, undissolved particles should be fished out and then be put under the microscope. They are fibrous in structure, cylindrical, and branching. As has been stated, the maximum temperature is soon attained. On the evening of the first day it may reach 104° Fahr. (axillary), and for several days it continues at about 103°, 104°, or even 105°, there being a slight morning remission and evening exacerbation. The fever pursues this course with little variation in favorable cases, until the period of crisis, when just before the defervescence a rise may take place. This rise in temperature in anticipation of the crisis is usual but by no means invariable. The pulse during the stage of hyperæmia is about 100—full, hard, and strong ; but, as consolidation takes place, if extensive or extending widely, a change occurs in the pulse ; it becomes less full, and, when the ischæmia of the arterial side has reached the lowest point, the pulse is small, soft, and weak, and the superficial veins are abnormally full and prominent. The skin, during the time of greatest fever, is mordicant, or burning-hot, and is dry or

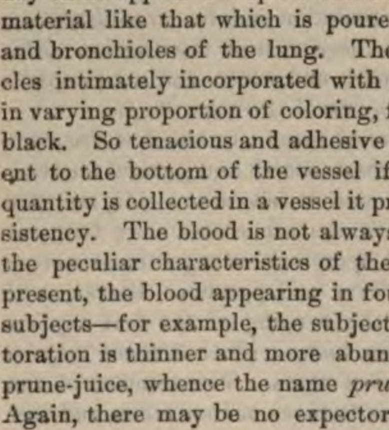

Fig. 29.—Fibrous Tissue in Sputa. (Beale.)

covered with a warm perspiration. If the skin is relaxed, dusky, cool, and covered with a cold sweat, the condition is unfavorable.

If the inflamed area is deeply situated and surrounded by healthy lung-tissue, the reactions produced on palpation and percussion are modified. On palpation the resistance is increased if the inflamed lung is exterior; not affected, if within. The vocal fremitus is somewhat increased. The sonority is diminished when the lung is consolidated; it is exaggerated when there is a layer of lung-tissue containing air overlying a consolidated area. Again, the sonority is exaggerated, or tympanitic, when in the beginning of the inflammation the lung still contains some air. The sound continues somewhat tympanitic in quality about the consolidated portion of the lung at the maximum. With the progress of the exudation, and when the peripheral portion of the lung is involved, there is greatly increased resistance, and the percussion-note over the inflamed area is flat, with still something of the tympanitic quality. The vesicular murmur becomes more and more feeble as the air less and less distends the alveoli. Within twenty-four to thirty-six hours there is heard, with or at the end of inspiration, a fine crackling sound over the region inflamed—the *crepitant râle.* This is wrongly said to be pathognomonic, since it occurs in acute tuberculosis, œdema of the lungs, etc.; but it is highly significant in that it is audible in so few conditions, and occurs in pneumonia over a restricted area. This *râle* has been compared to the sound produced by rubbing a lock of hair between the fingers in front of the ear, to the burning of some grains of salt on live coals, but it is most perfectly imitated by the crackling made by India-rubber sponge when pressed and allowed to expand in front of the ear. As the sound is produced by the separation of the bronchioles and alveoli, adherent by the viscidity of the albuminous exudation, it is obvious that it can occur only during inspiration. When consolidation takes place, the crepitant *râle* ceases, but can be heard in the neighboring parts of the lung undergoing the same process. Again, it becomes audible when the stage of resolution is reached. It is then known as *crepitatio redux*, but it then differs somewhat in quality, and is coarser and louder. The crepitant *râle* in children and old subjects is much like the crepitation redux. This *râle* is audible for a brief period only, during the stages of engorgement and exudation; presently the vesicular murmur ceases altogether; the respiration becomes sibilant, then blowing, and on the third day bronchial breathing and bronchial voice come on. The conductivity of the lung being increased by consolidation, the sound produced by the vibration of a column of air in the larger bronchi is communicated directly to the ear—whence the term bronchial breathing. The voice-sounds are communicated with equal distinctness to the ear from the larger bronchi—whence bronchial voice. When the lung-tissue is consolidated, the disease is at its maximum; there may be an extension of the area of inflammation in all directions, but the symptoms continue with uniform intensity for several days. We must now return to the rational symptoms and follow their development up to the period of crisis. The fever continues pretty uniformly at the point already mentioned, 102°, 103°, 104°, or 105°—there being a morning remission of less than a degree. The pain in the side lessens or ceases altogether. The decubitus is toward the right with the body flexed, so as to relax the muscles of the affected side, and thus take the pressure off; but the dyspnœa is less, because, the pain having declined, the respiration is free, but there is still some difficulty in respiration. The cough is more or less troublesome, and the characteristic rusty expectoration, or the more abundant "prune-juice," is brought up with every effort. Sometimes the expectoration is hæmorrhagic, and several ounces may be discharged at a time. The smallness of the pulse and feebleness of the cardiac impulsion are due to ischæmia of the arterial side, as has been pointed out; on the other hand, this state of the circulation may be largely due to depression of the forces. If the area involved in the inflammation is not very large, the pulse may continue full and strong up to the crisis; if this area is large and extending, then the fullness of the venous system and the emptiness of the arterial will have the effect just stated over the circulatory system; consequently, the condition of the circulatory system will afford valuable information in respect to the extent of lung-tissue involved in inflammation. A rapid and weak pulse—120, 130, 140—irregularities in the rhythm, and unequal filling of the artery, are very ugly symptoms, denoting cardiac failure. Delirium is a result of the diminished arterial supply and the venous stasis of the brain; there may be merely hallucinations or illusions, or noisy and violent delirium. Mental disturbance is more especially present in the cases of pneumonia occurring in drunkards; delirium tremens too often masks so completely the pulmonary symptoms that they are overlooked. In such cases, the pneumonia is the disease, and the delirium tremens the symptom or complication, instead of the reverse. The obstruction at the lungs and the consequent venous stasis affect other organs besides the brain. The liver is congested, and jaundice, more or less decided, is present in many cases, whence the name bilious pneumonia. Again, the pneumonia of malarious regions is so often modified by malarial infection that the biliary disturbance may be either caused or increased by this influence. Furthermore, an accompanying gastro-duodenal catarrh may, by an extension of the catarrhal process to the bile-ducts, set up a catarrhal jaundice. All of these influences coinciding, the biliary disturbance may enter largely into the symptomatology and therapeutics of the case. Very rarely a case of pneumonia may be complicated by acute yellow atrophy. The urinary secretion is altered in quantity and in composition ; the quantity is reduced ; the urea and uric acid are increased, and the chlorides are much diminished or disappear entirely. The chlorides are diverted to the inflamed part and from the urine, so that the return of the chlorides (chloride of sodium chiefly) to the urine signifies the cessation of the inflammation. So sensitive is this indication, that the return of the chlorides to the urine may precede for some hours the physical and rational signs which indicate the beginning of resolution. In consequence of the venous stasis, the hyperæmia of the kidneys may induce albuminuria, and the urine may contain also cast-off epithelium of the tubules, but the albuminuria is a transient state. It should be noted also that, during albuminuria, pneumonia arises as a complication, and not unfrequently a fatal one.

Pneumonia is one of the few diseases terminating by crisis. The critical phenomena consist in a sudden decline of temperature by crisis or lysis, and the occurrence of some special evacuation, as a large urinary discharge, a profuse diarrhœa, general sweating, an herpetic eruption, or considerable expectoration. The return in a few hours to the normal temperature or below it is the most conspicuous of these phenomena. As has been narrated, just before the defervescence, the temperature may rise higher than it had been, and the aspect of the case appear more formidable ; then the decline begins, and within twelve hours the normal or somewhat below is reached, or, if by lysis, the descent to normal occupies two or three days. The change thus wrought in the aspect of the patient is most remarkable. The countenance clears up, the difficulty of breathing subsides, the pulse falls to seventy, to sixty, even to forty per minute, and an herpetic eruption appears on the lips ; appetite returns, the skin is covered with warm perspriation, the urine increases in amount, the chlorides reappear, and the patient experiences an internal sense of well-being. The physical are in accord with these rational signs : moist sounds now appear in the bronchical tubes, and the sputa become lighter in color, and

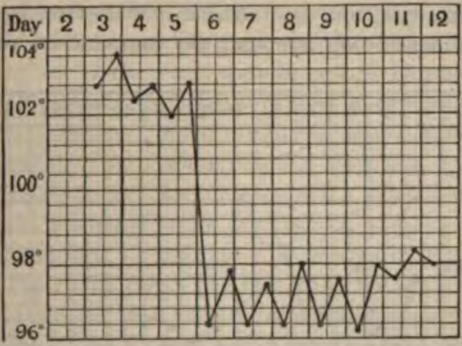

FIG. 30.—Temperature of Uncomplicated Pneumonia of Right Lung. Termination by Crisis.

an abundant expectoration of grayish-yellow muco-pus takes the place of the rusty sputa; *crepitatio redux*, coarser than *crepitatio indux*, appears along the outer border of the consolidated area; bronchophony is succeeded by a softer blowing sound; the flatness is now dullness, with more of the tympanitic quality, and the vocal fremitus is less decided. Careful examination of the sputa during the stage of resolution will disclose the presence of the fibrinous casts of the finer tubes, already described, and small masses, remains of the coagulated exudation in the air-sacs. The alveoli are gradually opened up to the admission of air, and under favorable circumstances the restoration of the lung is complete in a few days. In some unhealthy subjects, the victims of a diathesis, and sometimes those whose vital forces have been reduced by depressing treatment, repair is incomplete, and the affected part lapses into the chronic state. When the course is not toward crisis and health, there may be abortive attempts at crisis; there may be some considerable subsidence of the temperature, an illusive appearance of a critical evacuation in the way of an exhausting diarrhœa, for example, but the natural powers are not equal to the effort; there is no real improvement, the temperature rises even higher than before, and all of the symptoms develop new severity. The pulse declines in strength and volume and becomes very frequent, the dyspnœa increases, and an adynamic state, in which the tongue is dry, the face cyanosed, the breathing quick and shallow, and the debility great, supervenes. If delirium had existed before, it now assumes more of the low-muttering character; if it had not existed before, it is now apt to come on in the form of hallucinations; there are increas-

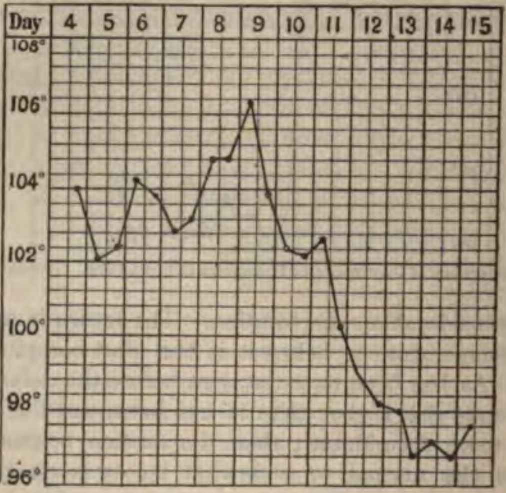

FIG. 31.—Temperature of Uncomplicated Pneumonia terminating by Lysis.

ing somnolence and a tendency to coma as the venous stasis and carbonic-acid poisoning increase, and finally a condition of more or less profound coma ushers in death.

Complications.—Pleurisy is a frequent complication, the two diseases occurring together in from ten to twenty per cent. A more acute pain and the usual signs of effusion are the only evidences of the existence of pleuro-pneumonia. The effusion must amount to six ounces to be detected with certainty (Juergensen). If there be extensive consolidation, the effusion must be proportionally small. Pleuritis is ascertainable with certainty only if there be sufficient effusion to displace the heart. The existence of pleuritis does not modify the course and behavior of the pneumonia itself, but the situation is rendered more grave by the simultaneous development of the two diseases. Capillary bronchitis is a very dangerous complication of croupous pneumonia, and may so conceal the latter as to appear as a case of catarrhal pneumonia. Emphysema is an occasional complication; it should be stated, however, that pneumonia is an ordinary mode of termination of emphysema. Pericarditis is more frequently a complication of pleuritis, but it may also occur in the course of pneumonia. Granular degeneration of the heart-muscle occurs in pneumonia when the temperature is persistently high, and is a serious complication. The occurrence of jaundice has been alluded to as a symptom, and its mechanism explained. That pneumonia is a disease of great frequency and fatality in malarious regions is undoubted. Rheumatism and gout are also frequently associated with pneumonia, and to these may be added acute alcoholismus. Pneumonia of diathetic origin is severe or not according to the character of the diathesis; it is very fatal in the alcoholic, but not more so than the uncomplicated malady in the rheumatic or gouty form. The existence of a *typhoid pneumonia* is pretty generally admitted, but on questionable evidence. Pneumonia is an occasional complication of typhoid fever, but it is not a typhoid pneumonia. This term is applied to a form of pneumonia occurring in the weak and debilitated, and has therefore a specially adynamic character. There is not the fever process which we designate typhoid; there exists a pneumonia to which a specially adynamic character has been imparted by the depressed state of the vital forces. The term has been so far generalized that, in many places, every severe case of pneumonia is called typhoid pneumonia.

Course, Duration, and Termination.—Croupous pneumonia is a well-defined, self-limited disease, which passes through its several stages with considerable uniformity. The stage of congestion or engorgement occupies the first twenty-four to thirty-six hours; the stage of exudation or red hepatization—that period occupied by the pouring out and coagulation of the exudation—continues up to the crisis, which marks the beginning of the next stage. The crisis in pneumonia occurs somewhere from the fifth to the eleventh day of the disease, so that the exudation stage lasts from two to eleven days. The stage of resolution begins with the phenomena of the crisis, and lasts two to four days till convalescence is established. In rare cases (abortive forms) critical phenomena may occur even earlier than the fifth day. In the largest number the crisis begins on the seventh day, and, according to Traube, always on the odd days, reckoning from the day of the initial chill, but if we except the seventh day the statement of Traube must be denied. The stage of purulent transformation is not distinctly separated from the stage of exudation or red hepatization, unless the occurrence of an abortive attempt at crisis fixes the period. It begins about the middle of the second week, and continues for several days to a week. The whole course of pneumonia is therefore comprehended within three weeks, but favorable cases may terminate in two weeks. The mortality from pneumonia has been and continues to be a subject of warm discussion on the part of those who advocate some special plan of treatment. Accuracy in diagnosis and skill in treatment are such uncertain elements in the statistics of mortality, under different plans of treatment, that but little reliance can be placed on the statistical method as applied to therapeutical questions. According to the most approved of the modern methods, the mortality ranges from five to twenty-five per cent. In determining a fatal result in croupous pneumonia, so much depends on the condition of the individual attacked, or the diathesis with which his system is tinctured, that no comparison of systems of treatment can be accurate that does not take note of them. Death is usually due to collapse—that is, cardiac failure, and obtunding of the nervous centers. This state is not necessarily caused by purulent transformation—it may be due to failure of heart, and lungs, and brain, before the end of the stage of red hepatization. Death may be caused by the mere extent of the lesions in the lungs, inducing asphyxia; these lesions consisting not only of localized pneumonia, but also of collateral hyperæmia and œdema. The effects of the pulmonary changes are enhanced by the stasis in the cerebral veins and ischæmia of the arteries, and by cardiac paresis. In subjects extremely delibitated, the tissues in a scorbutic state, the termination may be by gangrene, but this is extremely rare. The formation of an abscess is also rare, but is more common than gangrene. An example of encysted abscess which had been carried many months has been mentioned; usually the abscess formed during the stage of gray hepatization terminates in a short time by discharge either into the pleural cavity or into a bronchus. The presence of a quantity of the elastic tissue of the lungs in the sputa and the occurrence of repeated rigors and profuse sweats indicate the formation of the abscess. If it become encysted, just as is the case in abscess in the liver or in the brain, the acute symptoms subside, the fever falls, the rigors and sweats cease, but yet some unfavorable symptoms continue—there are cough, fever, dry tongue, emaciation, and weakness, and the appropriate physical signs. In a variable period the abscess terminates in some of the modes already described. The termination may be in the chronic form. There are then no critical phenomena; the fever gradually diminishes, but does not cease; the difficulty of breathing lessens, but there is more or less embarrassment on making any effort; the cough also continues, and muco-pus and fibrous tissue are expectorated; the weakness and emaciation do not improve if the decline does not go on, and the physical signs of condensation of the pulmonary tissue remain. The subsequent behavior is influenced by the local condition and the direction taken by the products of inflammation. There may ensue a gradual liquefaction of the exudation, its softening and extrusion may be effected without much damage to the pulmonary parenchyma, and after some months a cure be effected. On the other hand, the exudation may undergo caseation, with the usual history of pulmonary consumption. The caseation of the inflammatory products of croupous pneumonia is held to be doubtful by many, and is not regarded as common. The clinical history is that of caseous pneumonia, and need not be discussed until that subject is reached. Finally, death may be caused by one of the complications, as pericarditis.

Diagnosis.—Ordinary well-defined cases are recognized without difficulty; it is the obscure or anomalous forms that occasion mistake. Pleurisy with effusion is very frequently confounded with pneumonia. They are differentiated by the following points: The onset of pneumonia is sudden, by a rigor, and followed by a high temperature—pleurisy begins more gradually, there is chilliness for a day or two, and the rise of temperature is gradual; in pneumonia, the pain is rather dull, or a feeling of soreness diffused over a considerable space—in pleurisy, a sharp stitch, which can be covered by a finger; in pneumonia, there is audible, on inspiration only, a crackling sound, the crepitant *râle*—in pleurisy, the friction-sound, synchronous with the respiratory movements; in pneumonia, the crepitant *râle* is succeeded by bronchophony, which continues—in pleurisy, when the effusion partly compresses the lung, a modified bronchophony, but, when the lung collapses, all voice and breath sounds cease; in pneumonia, the dullness has a tympanitic quality, and is fixed in position—in pleurisy, the dullness is flat, and changes with the gravitation of the fluid; in pneumonia, the organs retain their position—in pleurisy, the heart is pushed aside and the liver downward by the effusion; pneumonia is self-limited, and terminates by crisis—these phenomena are wanting in pleurisy, the duration of which is indefinite; subsequent to the crisis, the behavior of the two diseases is so different that further comparison is unnecessary. Next to pleuritis with effusion, pneumonia is confounded with catarrhal pneumonia. They differ in onset

—pneumonia sudden, with a rigor, and pain in the side—catarrhal pneumonia with an ordinary bronchitis, and a feeling of soreness rather than pain under the sternum; pneumonia, as a rule, is unilateral, self-limited, terminating by crisis, or ceasing within three weeks —catarrhal pneumonia is bilateral, not limited nor terminating by crisis, and indefinite in duration; if double, which is rare, pneumonia is limited to a portion of either lung, while catarrhal pneumonia is diffused over both. The differentiation of bronchitis and croupous pneumonia rests upon the same points. In respect to physical signs, the differences are marked: In pneumonia, the vocal fremitus is increased, and there is increased resistance on palpation—in bronchitis, the vocal fremitus is unaffected, and there is no change in the resistance; in pneumonia, there is dullness on percussion—in bronchitis, the percussion-note is unaltered; in pneumonia, on auscultation, there is audible the crepitant *râle*, which disappears and is replaced by bronchophony—in bronchitis, there is no crepitant but a sub-crepitant *râle*, followed, not by bronchophony, but by sub-mucous and mucous *râles*. The *râles* in pneumonia or the bronchophony are audible at the seat of inflammation only—in bronchitis, they are diffused over the chest. An uncomplicated pneumonia differs from a pleuro-pneumonia in the following particulars: In pleuro-pneumonia there is more acute pain, a friction murmur as well as a crepitant *râle*, displacement of the heart and of other organs by the fluid, more absolute dullness on percussion, and less of the tympanitic quality to the percussion-note. Cases of pneumonia with cerebral symptoms may be mistaken for meningitis, but this can only happen should the chest not be examined. In pneumonia of the aged, and, in some cases, in subjects of delirium tremens, there may be no cough or other rational symptom to direct attention to the chest.

Treatment.—The constitutional tendencies, the actual state, and the surrounding circumstances should receive careful attention in deciding on a plan of treatment. A vigorous, healthy subject, free from constitutional vice, will require and bear a more vigorous handling than a broken-down alcoholic. If seen at the beginning, during the stage of congestion, there can be no doubt as to the good effects of venesection or the local abstraction of blood. The author, for the most part, has not been in favor of blood-letting, but his experience has taught him that systemic or local bleeding may be of the highest service in cases of pneumonia. He has seen no cases of late years that could not bear from five to ten full-sized leeches, and many that were improved by venesection of two to eight ounces. Whether or not the abstraction of blood being decided on, the next question is the use of calomel. On this point, also, the author's opinions have been modified by more extended observation. No cases of pneumonia are seen in these latitudes that are not accompanied by considerable hepatic disorder. There is a more intimate relation between hepatic disorder and pneumonia, I believe, than has been made out as yet. A high degree of overirritation and a temporary increase of productivity is a nearly constant or quite constant condition of the liver in pneumonia. The external evidences are the intense yellowness, or the jaundiced hue of the skin of the conjunctivæ and elsewhere. If, instead of overaction, it is a disturbance in which excretion from the pancreas, liver, and intestinal glands is the cause, the indications afforded thereby are equally in the direction for the action of calomel. Modern researches have shown that calomel has a sedative action on the liver, lessens bile production, and probably the whole functional activity of the liver. It may be that, as the blood in the hepatic veins is so much higher in temperature than in the portal, the reduction in the febrile heat is due to the lessened functional activity of the liver. It is clear that calomel has an antipyretic action. Hence, at the outset, it should be given in a dose of from three to five grains. The repetition of the dose depends on the result of the first; but as a rule from two to four doses are required—on the first, second, and third days of the actual inflammation.

In cases accompanied by depression, or with light sthenic reaction, a few cups or leeches may be applied, and small and frequently repeated doses of the tincture of aconite-root (two to five drops every two hours) may be given. A large mustard poultice should be put on the chest, and removed when the skin is reddened, to obtain its stimulant effect on the vaso-motor nerves within, and the feet should be immersed in a hot mustard foot-bath. If the viscid secretion is pouring out in the air-sacs and bronchioles, and coagulating, it is necessary to use some agent which possesses the power to lessen the viscidity and coagulation. Hughes Bennett employed the potassa salts (liquor potassii citratis) or an extemporaneous solution of the bicarbonate, and his results were admirable. Ammonia, originally suggested by Richardson, has been latterly used more freely than potassa, and, as the author believes, with better results. Probably the most advantageous method of administering it is the solution of the carbonate in liquor ammonii acetatis (℥ ss.—gr. v to x) every three or four hours. By the German school the muriate is preferred in corresponding doses, but it does not appear to the author to be so useful. The ammonia solution should be continued up to the crisis. As soon as consolidation of the lung is accomplished, all arterial sedatives of every kind should be discontinued.

Assuming that pneumonia is a specific disease, like typhoid, Juergensen maintains the necessity for the use of antipyretics, among which he places the cold bath first; and the success of his treatment certainly seems to justify his theory. He demonstrates that there is no danger in putting a pneumonic patient in a bath, and that the reduction of temperature by it exercises a favorable influence over the progress of the disease. Next to the bath, quinine is most useful as an agent for reducing fever, but it must be given in scruple doses every four hours until the temperature falls to a proper point, when it may be suspended until the temperature rises again in twenty-four to thirty-six hours. The new antipyretic—antipyrin—has proved to be so certain and powerful a means of reducing febrile heat, that it may be resorted to in cases where the temperature approaches the condition of hyperpyrexia, or instead of quinine as suggested above for this purpose.

The author feels it necessary to emphasize the evil effects of cardiac sedatives during the stage of exudation and of coagulation of the exudate. The administration of veratrum viride, digitalis, aconite, and tartar emetic, can only add to the burden of the heart, already laboring in consequence of the stasis on the venous side, and lack of blood on the arterial side. Paralysis of the heart is one of the most imminent dangers, because of this state. It is true that a continued high temperature contributes to bring about paralysis of the heart, but we possess the means of correcting this by the administration of quinine, and by cold baths or the cold wet pack. While arterial and cardiac sedatives are to be avoided at the stage of red hepatization, it is necessary also to avoid the immoderate use of alcoholic stimulants. These are needed, and in full doses in inebriates at the period of crisis, and when the stage of purulent transformation is reached if there are a rapid and weak pulse, a relaxed and clammy skin, and delirium. Protracted wakefulness and delirium need careful management. Opium or morphine must be avoided, owing to the state of the pulmonary circulation, and the collateral hyperæmia and œdema. Then it is that chloral hydrate serves a most useful purpose; it procures sleep, quiets delirium, and has a good effect on the exudation. Care must be exercised, for large or frequently repeated doses may cause paralysis of the heart; fifteen grains at night, with ten more in two or four hours, if the first dose is insufficient, is all that is required usually. Aliment must be carefully administered from the beginning, without waiting for depression to come on. Beef-juice, milk, egg-flip, wine-whey, chicken or mutton broth, etc., should be systematically administered every three hours. In weak subjects, a little wine may be given from the beginning. As already stated, the pneumonia of the inebriate requires alcoholic stimulants from the first symptom—for the delirium accompanying it is due largely to the sudden withdrawal of the supply, or the inability to retain it. Much has been said about the blistering-point in pneumonia. Counter-irritation is useful during the stage of congestion, as already indicated, but a fugitive counter-irritant, as a mustard-plaster, is all that can be properly used. When the crisis occurs, a blister is very useful. During the stage of red hepatization, turpentine-stupes, cotton wadding, or a flannel jacket, is useful unless the temperature is very high, when they do mischief. Flying-blisters are serviceable in promoting absorption, when resolution is imperfect and exudations still linger at the site of inflammation. To facilitate absorption in chronic, succeeding to acute pneumonia, the iodide of ammonium is highly beneficial. It may be administered with the iodide of iron, and in conjunction with the hypophosphites. If there are "prune-juice" expectoration, weak pulse, relaxed and sweating skin, turpentine in small doses, or eucalyptol, is extremely useful. During gray hepatization, they may be given for the double purpose of acting on the organ by which they are eliminated, and as cardiac stimulants.

EMBOLIC PNEUMONIA—PNEUMONIA FROM EMBOLISM.

Definition.—By *embolic pneumonia* is meant an infarction of the lung, due to embolic blocking of a vessel.

Causes.—From the right cavities of the heart, or from some part of the venous system, an embolus is dislodged, and, entering the current of the blood, is deposited in a branch of the pulmonary artery. The circumstances under which clots form in the right cavities of the heart have been set forth elsewhere.

Pathological Anatomy.*—The emboli which give rise to embolic pneumonia are of two kinds, simple or non-infective and infective. The former act in a merely mechanical manner by closing the vessels and preventing the passage of blood to the parts supplied by them; the latter not only obstruct vessels like the former, but the infective material contained in them sets up a local infectious process. The size of the embolus, and consequently the capacity of the vessel obstructed, varies considerably, the resulting infarction being from a pea to a hen's-egg in size. If a simple embolus, the damage is confined to the area occupied by the infarction; but, if an infective embolus, a suppurative inflammation arises and an abscess is the result. To the formation of an infarction it is necessary that the embolus lodge in a terminal artery of Cohnheim—an artery without anastomoses—for, if the obstructed artery is connected by branches with others, the circulation in the obstructed area may be restored through collateral channels. If the obstructed artery be a terminal one, as are those of the outer part of the lung in a restricted sense, the pressure in the veins causes a gradual filling of the obstructed vessels through the capillaries. Now, as the walls of these obstructed vessels are not properly nourished by the blood thus in a state of stasis, the blood diffuses through into the surrounding textures, which constitutes the infarction. Such an infarction is not often possible at the root of the lung, for here the anastomoses are too numerous, although they do sometimes occur;

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* In the account of this process, Cohnheim's classical work, "Untersuchungen ueber die embolischen Processe," Berlin, 1872, Hirschwald, is followed.

but it is at the periphery that they usually form. As the vessels proceeding from the root of the lung toward the periphery divide dichotomously, it is obvious that, when an embolus obstructs one, the resulting infarction must be wedge-shaped—the base of the wedge being toward the periphery of the lung, or outwardly. If a section be made through an infarction, its outline will be seen rather sharply defined, its color of a deep blood-red, and it will exude blood on slight pressure. If it has been formed for some time, its structure is denser from an infiltration of the alveoli, whence it presents a granular appearance; it is dark-brownish in color, is drier, and exudes but little blood, and is very friable, easily breaking up into a pulverulent mass. The bronchi contain a frothy, bloody fluid. The tissue of the lung about the infarction becomes hyperæmic and œdematous. The pleura overlying it is deeply congested, or it may be inflamed and coated with a firmly adherent albuminous exudation, while the cavity contains more or less bloody serum. The infarction undergoes various changes; the blood is gradually transformed, becomes fatty, and is absorbed, although patches of altered hæmatin remain; the proper tissue of the lung undergoes atrophy, the connective tissue multiplies, and in this way a cure is effected, the lung being rendered useless to the extent of the infarction. In other cases an embolic abscess is produced, the embolus being infective; but it does not have a wedge-shape; it is globular, and presents the appearance of an ordinary purulent collection. In rare cases an infarction becomes gangrenous. Infarctions are found more frequently in the right lung.

Symptoms.—As the embolus proceeds most frequently from the right side of the heart, the clinical history is that of some cardiac disease; but it may be produced in some distant part of the venous system under circumstances which favor thrombosis. The prominence and urgency of the symptoms will depend on the size of the infarction. If it be small in extent, there may be no disturbance; even if quite large, the symptoms may be masked by the coexistent disease. If a large branch of the pulmonary artery be suddenly closed, there will be acute dyspnœa of extreme severity, the patient will gasp for breath, become deeply cyanosed in a few minutes, and, may be, die at once. Sudden difficulty of breathing is the most significant symptom at the time of lodgment of the embolus, especially if there is nothing in the condition of the heart to account for the dyspnœa. Fever comes on some days after the obstruction, but the rise of temperature is not very great. There may be chills, but they are not constant, except in the case of pyæmia. Bloody expectoration appears in a few days after the initial dyspnœa, and is usually inconsiderable in quantity. Besides blood, there is a viscid mucus which is the body of the sputa, and, as it adheres rather tenaciously, a good deal of coughing is necessary to bring it up. Pain begins with the implication of the pleura, and has the usual characteristics of pleuritic pain: it is acute and lancinating, and is increased by the movements of respiration. There are present the usual physical signs of consolidated lung—dullness on percussion, bronchial voice, and bronchial breathing. There may be a friction-sound due to the pleuritis, and also the evidences of effusion into the pleural cavity. It is obvious that the diagnosis of embolic pneumonia is difficult and uncertain. The sudden occurrence of dyspnœa, followed by bloody expectoration continuing eight or ten days, and the evidences of consolidation, are the only symptoms to indicate the real nature of the malady. If the history furnished the source of the embolus, the diagnosis would be proportionally facilitated. The prognosis is generally unfavorable, notwithstanding small infarctions may get well. There is no plan of treatment which can affect a mechanical condition of this kind, unless ammonia may dissolve an embolus. This should be tried.

CATARRHAL PNEUMONIA.

Definition.—Various terms have been applied to this disease, as *capillary bronchitis*, *lobular pneumonia*, *broncho-pneumonia*, etc. As right views with regard to it are necessary to a proper conception of pulmonary consumption, it is discussed here somewhat in advance of its proper position. By the term *catarrhal pneumonia* is meant a catarrhal inflammation involving the bronchioles and alveoli. It may be *acute* or *chronic*.

Causes.—Catarrhal pneumonia may be an extension downward of a catarrhal process beginning in the bronchial tubes. It is probable that a catarrhal inflammation never begins, under any circumstances, in the alveoli. Typical examples of this disease occur during certain of the exanthemata, notably measles and whooping-cough. It is intimately associated with certain diatheses, as rickets and scrofula, and with structural alterations of the heart and lungs, as mitral lesions and emphysema. It is frequent in early life and in old age, and is less so at the period of greatest bodily vigor. Bad hygienic influences as to dress, habitations, humidity, and exposure, favor its development. Climate is an important factor, and the period of most extreme variations is the period of greatest prevalence of this disease.

Symptoms.—The acute form is the type; the chronic differs from it merely in duration and severity of the symptoms.

The initial symptoms are chilliness followed by fever, soreness of the chest, chiefly beneath the sternum, cough, and expectoration of a frothy mucus, and some difficulty of breathing. These symptoms in the acute form of the disease quickly develop into the more serious and characteristic proper to catarrh of the finer bronchial tubes. An abundant secretion, poured out all along the bronchial tree, must greatly affect the functions of the lungs. The breathing soon becomes rapid, superficial, and labored, the accessory muscles of respiration are brought into play, and the alæ of the nose work quickly and continuously; the face is at first flushed and rather animated, and the eyes have a glaring expression, but the lips soon become bluish and cyanosis spreads over the face. The cough in the first onset is rather loud and bronchial, but, as the finer tubes become involved, it has more of a stridulous, husky character, and is often suppressed and partial because the difficulty of breathing is too great to permit the necessary expansion of the chest. The cough is also painful, and in children is attended with moans and crying, and they make attempts to restrain it because of the soreness in the chest. The fever soon rises to the maximum of 104° to 105°, and is nearly continuous, there being a slight morning remission. As the difficulty of breathing develops, there is increasing restlessness, never a moment of quiet, the struggle for breath and the search for an easier position being incessant. At first there are brief snatches of uneasy sleep, but, as the dyspnœa increases, a state of somnolence comes on which gradually deepens into coma, so profound at length that cough is suppressed. This somnolence is due to the deficient aëration of the blood and the accumulation of carbonic acid. Finally, the blood becomes wholly venous. Then the flush disappears from the face and is replaced by a death-like pallor, the cyanosis deepens about the lips, blue spots appear on the cheeks, and the superficial veins grow into thick black cords. The struggle for breath continuing, while the carbonic-acid poisoning increases, the most frantic but largely automatic efforts are made to remove supposed obstructions, and the patient, a child, may tear its skin about the neck and face with its nails, in the vain effort to remove them. On inspection, the cervical and other muscles auxiliary are seen actively engaged, and a deep depression of the abdomen from retraction of the lower ribs is made with every strong inspiration. On palpation, the vocal fremitus will be unaffected during the first few days, but, when the lobules have collapsed in considerable numbers, the physical conditions are changed, and the vocal fremitus will then be increased. On auscultation, *râles* are abundant all over the chest; they consist of sub-crepitant *râles*, which are somewhat coarser and louder than the crepitant, and are audible with both inspiration and expiration. With these also occur mucous and sub-mucous *râles*, produced in the larger tubes. The respiratory murmur becomes more and more feeble as the condition of atelectasis is produced; and, when a number of lobules are thus affected, over them the respiratory murmur ceases to be audible, a blowing sound is substituted, and this passes into bronchial breathing and bronchophony as the pulmonary tissue becomes consolidated. On percussion there is no change until the atelectasis occurs; the sonority is diminished as the lobules collapse, until dullness is reached; but the dullness has much of the tympanitic quality, owing to the proximity of unobstructed alveoli. In making percussion in children, it is important to strike lightly, otherwise the primary bronchi and trachea will be thrown into vibration. The pulse-rate does not always correspond to the range of temperature; it is usually higher. The pulse ranges from 140 to 200 or more in children, while in the aged it may be but little accelerated. Protracted high temperature may induce changes—parenchymatous degeneration of the cardiac muscle. If, therefore, during the course of this disease the pulse becomes feeble, irregular, and very rapid, the condition of the heart is one to arouse great solicitude. The appetite is poor, vomiting often occurs, and diarrhœa is by no means infrequent. The embarrassment to breathing caused by the act of eating and swallowing induces young children to avoid eating solid food, although they will often drink greedily. Cerebral symptoms are present to a greater or less extent in all cases: there may be headache, hallucinations, muscular twitchings, even convulsions, and the coma of carbonic-acid poisoning. So closely do the nervous symptoms belonging to catarrhal pneumonia simulate those of tubercular meningitis that it may be exceedingly difficult to diagnosticate between them. In the chronic or, rather, subacute form of catarrhal pneumonia the development is slow, the fever of moderate intensity, and the difficulty of breathing not pronounced. If there has been an attack of acute bronchitis, or of whooping-cough with more or less extensive bronchitis, when the catarrhal pneumonia develops, the cough subsides, but the depression of the vital forces, the cyanosis, and the extreme emaciation, indicate the growth of the more serious lesions. When these cases tend toward a fatal termination, the grave symptoms just mentioned increase, and carbonic-acid poisoning comes on, death occurring in more or less profound coma. Some cases pursue a different course; after a protracted subacute period in which the pulmonary lesions begin, an acute attack arises, and then the subsequent behavior is that of an ordinary acute case, death occurring in coma. When they tend to recovery, there is a gradual improvement in all the symptoms: the cyanosis diminishes, the dyspnœa lessens, the appetite improves, and gradually the general health is in part restored, the lungs imperfectly repaired.

Pathological Anatomy.—The changes involve the bronchial tubes and the lungs. The mucous membrane is the seat of an hyperæmia from the larynx down, but it increases in severity downward, reaching the maximum at the most dependent part of the lungs. The vessels are so deeply injected that the mucous membrane is a dark red, and at various points there are extravasations. The finer tubes are filled with a quantity of yellowish, creamy, purulent fluid. On section of the lung, drops of this exudation, escaping from the tubes, look just like pus escaping from a small abscess, especially if the divided tube has undergone dilatation—a change which takes place in the more protracted cases. This pus is probably made up of the young cells derived by multiplication of the epithelium, but especially of the lymphoid cells which migrate from the vessels, and are found in the sub-mucous connective tissue, in the alveoli, and in the bronchioles. There are two opinions now entertained in respect to the cellular elements which crowd the alveoli, and as to the part taken by the pavement epithelium. Among others, Rindfleisch maintains that these cells are produced by the multiplication of the epithelium, and derived in part from the proliferation of the lymphoid cells ; others, again, notably Buhl, deny the participation of the epithelium, and maintain that the products of the catarrhal inflammation are drawn into the alveoli by a species of suction. Besides the changes in the mucous membrane, the bronchial tubes and intervening connective tissue take part. The bronchioles become dilated if they have been long subjected to the inflammation, and the connective tissue undergoes hyperplasia, attaining to very considerable development. The formation of the very viscid exudation which takes place at the beginning of the process and the swelling of the mucous membrane are important elements in the collapse of the lobules (atelectasis) which is a conspicuous result in the sum of pathological changes. The collapse of the lobules takes place before the alveoli which form them are crowded with the products of the catarrhal inflammation. The mechanism of the collapse is about as follows : In the strong efforts in coughing or in expiration, or both, the air is forced out through the swollen tubes ; and, when the air has passed, the surfaces are brought into contact, and are made to adhere tenaciously. All of the residual air is gradually expelled in this way ; but, in the efforts at inspiration, the force is insufficient to separate the adherent surfaces, and, as the pressure is immediately increased in the adjacent lobules, the collapsed lobule is also compressed. The collapsed lobules are easily recognized by their appearance, which is of a dark-blue or purplish-blue color ; they are much firmer, do not crepitate, because they contain no air, and exude but little blood on section. The extent to which this process is carried varies in different cases. It begins in the most dependent part of the lungs, and advances forward and upward, involving much, sometimes the whole, of the lower lobe. In some chronic cases the process takes place chiefly in the upper lobes. Collapse of some lobules, the pressure continuing the same, necessarily involves the dilatation of others, and in this way emphysema results, the anterior portions of the lungs being affected chiefly. Attacks of catarrhal pneumonia in early life, imperfect repair only taking place, have much to do with the subsequent development of emphysema. After the lobules have collapsed, for a short period they continue permeable to air and may be inflated. The change in color and density which occurs when the collapse is effected is often mistaken for inflammation—whence the term "lobular pneumonia." If the collapse continue, an inflammatory process is set up, similar to but not identical with that of croupous pneumonia, for it never becomes granular. The inflamed part becomes more solid, is of a dark-brown color, which terminates in grayish red; it begins in the center of the lobules and spreads outwardly; neighboring lobules affected in the same way coalesce, until ultimately a whole lobe may be involved. Then it presents to the eye, when the process is completed, a bluish-gray appearance; on section it is found to be homogeneous, very firm, and tough. Before this final stage is completed it is very friable. The purulent matter in the bonchi and the catarrhal products in the alveoli undergo the cheesy transformation. The subsequent history is that of "caseous pneumonia." Those portions of the pleura in contact with the inflamed lobules become hyperæmic, inflame, an exudation is poured out, and adhesions form, or effusion takes place in the thoracic cavity. Not every case tends to death, or to the chronic changes above described. Partial recovery ensues in a considerable number, complete recovery in but few. When the collapsed lobules inflame, unless there be but few, restoration seems hardly possible even in the sense of a partly useless lung. If the lobules are capable of being distended again with air, and the catarrhal inflammation subsides in the bronchioles and alveoli, a cure is then possible. The purulent contents of the bronchi are brought up by coughing, and swallowed or expectorated; the watery portion of the exudation in the alveoli is absorbed; the cells disintegrate, become granular and fatty, and are ultimately absorbed—thus restoring the alveoli to the admission of air. The fluid and the cells of the intervening connective tissue pass through the same process, and thus the injured part is restored, except that its elasticity continues impaired for a long time.

Complications and Sequelæ.—The complications are really parts of the malady in its entirety. Bronchitis is always present, and laryngitis frequently. Pleuritis is a necessary result when the peripheral portion of the lung is involved. The sequelæ are very important. As was indicated under the head of pathological anatomy, there are two diseases which result from catarrhal pneumonia—emphysema and caseous pneumonia. The former is a result of the atelectasis or collapse of the lobules; the latter is an outcome of the changes in the catarrhal products which crowd the alveoli, in the bronchi themselves, and in the intervening connective tissue. In the account to be presently given of these diseases, the course of development from one to the other will be set forth.

Course, Duration, and Termination.—The course of catarrhal pneumonia is from a catarrh of the larger tubes to a catarrh involving the ultimate bronchioles, and probably the alveoli. There are two principal phases in the subsequent course: the development of the catarrhal process; the collapse of the lobules, and the transformations which they undergo. Restoration may occur by a retrograde change in the catarrhal products and by absorption, and the collapsed lobules may be again expanded. Often the restoration is partial, and the lung may remain contracted and atrophied at the site of the collapsed lobules. In still other cases the bronchial tubes are dilated, the connective tissue undergoes hyperplasia and thickening, the catarrhal products become caseous, and the collapsed lobules slowly inflame. It is obvious that the duration of such a malady must be subject to great variations. The simplest case of catarrhal pneumonia can hardly be concluded in a less time than two or three weeks. In fatal cases, death may occur in a day or two or within a week. In rapidly fatal cases death is due to such a blocking of the bronchioles that the blood can not be aërated, death occurring in deep coma from carbonic-acid poisoning. In chronic cases death occurs in two modes: by an acute exacerbation; by gradual failure of the vital power, by the changes of catarrhal pneumonia, or the results of chronic inflammation in the collapsed lobules. In a large proportion of cases of catarrhal pneumonia in which recovery takes place, there is not a complete restoration, and hence the production of emphysema in after-years.

Prognosis.—About one half of the cases of catarrhal pneumonia prove fatal. The prognosis must be guarded, not only as respects immediate mortality, but the future prospects of such patients. The more acute the attack the greater the danger of a fatal result, for acuteness in the attack means the collapse of many lobules, and the more extensive the area of disease, the more formidable the case in every aspect. The younger the subject the more dangerous an acute attack is, or indeed any attack of catarrhal pneumonia. Diatheses play an important part in the prognosis, for scrofulous and rachitic subjects are less able to bear up under the inflammation. The prognosis is also much influenced by the bodily state, for the less the power of resistance the more severe the disease.

Diagnosis.—Catarrhal pneumonia may be confounded with bronchitis, croupous pneumonia, acute tuberculosis, and œdema of the lungs. From simple bronchitis, capillary bronchitis is separated by the size of the moist *râles*, by the dyspnœa in the one, its absence in the other; by the signs of consolidation of the lung-tissue in the one, by the absence of such consolidation in the other; and, finally, by the subsequent history so different in the two diseases. Croupous pneumonia is unilateral, or, when bilateral, limited to a certain area; catarrhal pneumonia is bilateral and diffused over both lungs. Besides the difference in the physical signs recapitulated under the head of croupous pneumonia, there is the remarkable difference in the behavior, one being a self-limited disease, the other having no fixed duration. Acute tuberculosis at its onset is characterized by the presence of a capillary bronchitis, so that a differentiation is possible only by a study of the clinical history and course of the two affections. Œdema of the lungs is accompanied by similar symptoms as regards the dyspnœa and the physical signs ; but œdema is not a feverish state, and it is accompanied by albuminuria or some evident cause.

Treatment.—The chief source of danger in catarrhal pneumonia is the universal presence of a viscid secretion, which interferes with the entrance of air and thus prevents proper oxygenation of the blood, and causes collapse of the lobules, indirectly. The agents most useful to diminish the viscidity and favor the excretion of the exudation are the preparations of ammonia. The author has obtained the best results from the carbonate (three to six grains) and the iodide of ammonia (four to eight grains) in solution every two hours. The muriate has been much prescribed for the same purpose, but the iodide and carbonate are more efficient. These should be perseveringly administered. If the symptoms are subacute, the oil of turpentine, eucalyptol, and copaiba are very active in checking the formation and favoring the extrusion of the exudation in the tubes. Of these, probably copaiba is the best, as it may be more energetically pushed than the others. These stimulating expectorants, as they are called, owe their efficacy chiefly to the fact that the volatile oil which they contain is eliminated by the lungs and acts locally. They may be used in the acute cases also, after the subsidence of the most acute symptoms, and at the same time that the ammonia preparations are administered. If there be excessive dyspnœa, notwithstanding the use of these remedies, the accumulated muco-pus must be dislodged by emetics. Apomorphine is the most efficient of the emetics, and can be administered in the way to secure the best effects—by hypodermatic injection. Great care must be exercised in the use of this remedy, since occasionally profound narcotism is produced by it, probably due to the presence of morphine. The author has used the subsulphate of mercury, with most excellent effect, as an emetic in catarrhal pneumonia. Although this is a poisonous substance, no danger need be apprehended from it, since it comes up with the vomited matters. It can be given in from two to three grains at a dose, rubbed up with some sugar. Besides its emetic action, the subsulphate seems to have the power to check the formation of the muco-pus. The repetition of the emetic depends on the state of the case—every few hours it may be administered if the dyspnœa and the cyanosis require it. The immediate result of the emetic action ought to be an improvement in the breathing and lessening of the cyanosis. If the fever is great and the arterial tension high, good results are obtained from the combined use of tincture of aconite-root and tincture of belladonna—two drops of the former and one drop of the latter to a child of two years, every two hours. Continued high temperature demands the use of quinine and digitalis. To a child of two years three grains of quinine and one fourth of a grain of digitalis can be given morning, noon, and even-they undergo. Restoration may occur by a retrograde change in the catarrhal products and by absorption, and the collapsed lobules may be again expanded. Often the restoration is partial, and the lung may remain contracted and atrophied at the site of the collapsed lobules. In still other cases the bronchial tubes are dilated, the connective tissue undergoes hyperplasia and thickening, the catarrhal products become caseous, and the collapsed lobules slowly inflame. It is obvious that the duration of such a malady must be subject to great variations. The simplest case of catarrhal pneumonia can hardly be concluded in a less time than two or three weeks. In fatal cases, death may occur in a day or two or within a week. In rapidly fatal cases death is due to such a blocking of the bronchioles that the blood can not be aërated, death occurring in deep coma from carbonic-acid poisoning. In chronic cases death occurs in two modes: by an acute exacerbation; by gradual failure of the vital power, by the changes of catarrhal pneumonia, or the results of chronic inflammation in the collapsed lobules. In a large proportion of cases of catarrhal pneumonia in which recovery takes place, there is not a complete restoration, and hence the production of emphysema in after-years.

Prognosis.—About one half of the cases of catarrhal pneumonia prove fatal. The prognosis must be guarded, not only as respects immediate mortality, but the future prospects of such patients. The more acute the attack the greater the danger of a fatal result, for acuteness in the attack means the collapse of many lobules, and the more extensive the area of disease, the more formidable the case in every aspect. The younger the subject the more dangerous an acute attack is, or indeed any attack of catarrhal pneumonia. Diatheses play an important part in the prognosis, for scrofulous and rachitic subjects are less able to bear up under the inflammation. The prognosis is also much influenced by the bodily state, for the less the power of resistance the more severe the disease.

Diagnosis.—Catarrhal pneumonia may be confounded with bronchitis, croupous pneumonia, acute tuberculosis, and œdema of the lungs. From simple bronchitis, capillary bronchitis is separated by the size of the moist *râles*, by the dyspnœa in the one, its absence in the other; by the signs of consolidation of the lung-tissue in the one, by the absence of such consolidation in the other; and, finally, by the subsequent history so different in the two diseases. Croupous pneumonia is unilateral, or, when bilateral, limited to a certain area; catarrhal pneumonia is bilateral and diffused over both lungs. Besides the difference in the physical signs recapitulated under the head of croupous pneumonia, there is the remarkable difference in the behavior, one being a self-limited disease, the other having no fixed duration. Acute tuberculosis at its onset is characterized by the presence of a capillary bronchitis, so that a differentiation is possible only by a study of the clinical history and course of the two affections. Œdema of the lungs is accompanied by similar symptoms as regards the dyspnœa and the physical signs ; but œdema is not a feverish state, and it is accompanied by albuminuria or some evident cause.

Treatment.—The chief source of danger in catarrhal pneumonia is the universal presence of a viscid secretion, which interferes with the entrance of air and thus prevents proper oxygenation of the blood, and causes collapse of the lobules, indirectly. The agents most useful to diminish the viscidity and favor the excretion of the exudation are the preparations of ammonia. The author has obtained the best results from the carbonate (three to six grains) and the iodide of ammonia (four to eight grains) in solution every two hours. The muriate has been much prescribed for the same purpose, but the iodide and carbonate are more efficient. These should be perseveringly administered. If the symptoms are subacute, the oil of turpentine, eucalyptol, and copaiba are very active in checking the formation and favoring the extrusion of the exudation in the tubes. Of these, probably copaiba is the best, as it may be more energetically pushed than the others. These stimulating expectorants, as they are called, owe their efficacy chiefly to the fact that the volatile oil which they contain is eliminated by the lungs and acts locally. They may be used in the acute cases also, after the subsidence of the most acute symptoms, and at the same time that the ammonia preparations are administered. If there be excessive dyspnœa, notwithstanding the use of these remedies, the accumulated muco-pus must be dislodged by emetics. Apomorphine is the most efficient of the emetics, and can be administered in the way to secure the best effects—by hypodermatic injection. Great care must be exercised in the use of this remedy, since occasionally profound narcotism is produced by it, probably due to the presence of morphine. The author has used the subsulphate of mercury, with most excellent effect, as an emetic in catarrhal pneumonia. Although this is a poisonous substance, no danger need be apprehended from it, since it comes up with the vomited matters. It can be given in from two to three grains at a dose, rubbed up with some sugar. Besides its emetic action, the subsulphate seems to have the power to check the formation of the muco-pus. The repetition of the emetic depends on the state of the case—every few hours it may be administered if the dyspnœa and the cyanosis require it. The immediate result of the emetic action ought to be an improvement in the breathing and lessening of the cyanosis. If the fever is great and the arterial tension high, good results are obtained from the combined use of tincture of aconite-root and tincture of belladonna—two drops of the former and one drop of the latter to a child of two years, every two hours. Continued high temperature demands the use of quinine and digitalis. To a child of two years three grains of quinine and one fourth of a grain of digitalis can be given morning, noon, and even-is no underlying constitutional state to invite other diseases; some pass into caseous pneumonia and phthisis, because they are tainted with the strumous diathesis; in a small number acute miliary tuberculosis develops. A strumous diathesis, not inherited, may be gradually acquired under the influence of bad hygiene—as living in a dark, damp, and foul habitation, with insufficient and improper food, and exhausted by overwork, anxiety, etc. If such influences are not sufficient to develop the strumous diathesis, at least they cause a bodily state in which caseation readily takes place in the inflammatory products of catarrhal pneumonia. Caseous phthisis is comparatively common in early life, because at this period measles, whooping-cough, and catarrhal pneumonia frequently occur. It may happen at any period, but is more common up to thirty-five than subsequently. As regards sex, the liability to this form of phthisis, it seems to the author, is greater in the female.

Pathological Anatomy.—In the description of the pulmonary lesions of catarrhal pneumonia, it was shown that the alveoli of the lungs are crowded with cells, and that the branchioles are filled with yellowish muco-pus. The part which the epithelium of the alveoli takes in these changes is disputed. According to Rindfleisch this pavement epithelium undergoes desquamation and other changes. "The cells first become looser, their attached surfaces are covered with a thick layer of finely granular protoplasm; at the same time in each cell the nucleus, which was before hardly visible, becomes swollen and is segmented. Thus are formed large granular epithelial cells, with rounded polygonal contours, and containing one or more nuclei." According to Buhl, the alveoli not containing a mucous membrane can not undergo the catarrhal process, and, therefore, the cells which so crowd the alveoli must be drawn or sucked into them. Besides the cellular elements filling the bronchioles and alveoli, an enormous infiltration of cells takes place into the intervening connective tissue—"many of them with two nuclei, nearly all with several surfaces, flattened." When this infiltration of cells has reached the point of distending the septa between the alveoli, the vessels are so compressed that the circulation in them is suspended. Hyperplasia of the connective tissue, although denied by Rindfleisch, does take place according to other investigators, and, in contracting, considerable shrinkage occurs, and a dense homogeneous mass results, made up of the distended alveoli, the infiltrated septa, the bronchioles dilated and filled with muco-pus and the contracting connective tissue, and is now in a condition preparatory to the cheesy transformation. The caseous change consists in absorption of the watery parts, the fatty degeneration of the cellular elements, and granular disintegration of the fibrinous material, so that ultimately a soft solid is produced, yellowish in color, and having the appearance of cheese. In the mass are inclosed all the pulmonary elements—the acini, the bronchioles, the vessels, etc. "These nodules are surrounded by atelectatic, œdematous, or gelatinous parenchyma in the preliminary stage of desquamative [catarrhal] pneumonia." The position of the catarrhal pneumonia resulting in the changes described is usually at the apex, but precisely the same alterations occur in other parts. They may result from a general catarrhal bronchitis which has subsided elsewhere, but usually the disease is of the subacute form already described in the previous section, and limited, as it has a great tendency to be, to the apices or to an apex. Sometimes a whole lobe, a whole lung (phthisis florida), becomes infiltrated, and undergoes the cheesy degeneration. The softening in these cheesy nodules or masses begins in the center, and consists at first of a central cavity and softened canals extending from the center to the periphery. According to Rindfleisch, the cheesy masses in the lumina of the bronchi are the first to soften, while that in the peribronchial and perivascular spaces resists the softening process for some time. The force exerted in respiration, the dilatation of the bronchi, and the contraction of the parenchyma of the lungs, are the agencies which procure extrusion of the detritus. Larger cavities are formed by the breaking down of the divisions between smaller ones. The shape, size, conformation, and appearance of cavities vary with their age. The admission of air sets up putrefactive changes, and, instead of an odorless, softened caseous matter, it becomes a foul, greenish, or grumous matter. When this is mixed with the sputa, elastic fibers are detected in it, and the yellowish-gray solid particles containing the bacillus, which are so characteristic a feature of the expectoration. At first, the interior of the cavity is irregular, rough, and is more or less full of disintegrating pulmonary tissue and projecting caseous material; but, when all this is discharged, it is smooth, and lined with a connective-tissue membrane, which furnishes a quantity of puriform fluid. If accumulation of the purulent con-

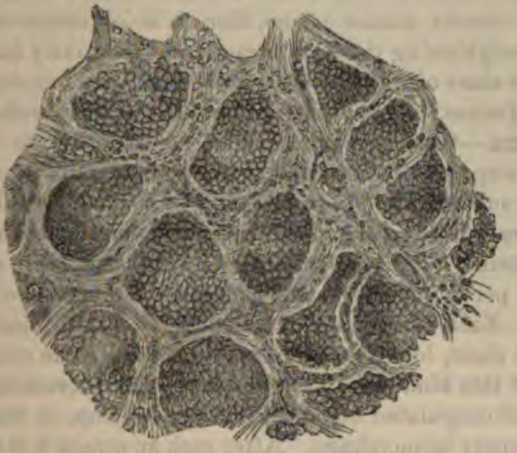

FIG. 82.—Caseous Pneumonia. (Thierfelder.)

tents of the cavity takes place, putrid decomposition occurs, and the pus becomes fetid. Hæmorrhage may be produced by erosion of a branch of the pulmonary artery. This accident would be much more common, if it were not that the vessels are early closed and cease to be pervious. In rare cases the mischief is confined to one or a few localities ; extrusion of the caseous matter occurs, there is no extension of the morbid process to neighboring tissue, contraction of the cavity takes place, and ultimately a mass of rather loose connective tissue remains to mark the site of the disease. This is the only mode of cure possible.

Symptoms.—Caseous phthisis does not conform to one mode of onset. As respects the initial symptoms, there are three types—the chronic, the subacute, and the acute, or *phthisis florida*. In the chronic form, the onset is so gradual that the first symptoms can not be fixed on with certainty. A susceptibility to colds has been observed, and gradually a persistent cough and expectoration of muco-pus are complained of. Each severe cold is accompanied by chilliness, some fever, pains in the chest, loss of appetite, and a troublesome cough. During an attack of this kind there may be bloody expectoration, or a mouthful or two of coagulated blood may be brought up, or there may be a smart pulmonary hæmorrhage. After such an attack it is observed that the "cold" does not get well ; that the cough and expectoration persist, that there are a daily morning chilliness, an evening fever, and a sweat some time during the night. A considerable loss of flesh is now observed, and there are great weakness and a feeling of exhaustion on slight exertion ; the appetite is poor, digestion is feeble, and, if a female, the catamenia are becoming scanty. In the subacute variety the onset is not so gradual. There is a history of a severe cold, with pain in the chest, a considerable fever, a troublesome cough, and abundant expectoration. The attack is severe enough to require confinement to bed for a few days, and, although after a week or two some improvement slowly takes place, and the patient gets about again, the symptoms continue ; there are fever, some sweating at night, a persistent cough, pains in the chest, expectoration at first of frothy mucus, then of muco-pus ; emaciation goes on and the strength does not improve ; the appetite is indifferent. In a portion of these cases, after the catarrhal products have become caseous, there is a period of comparative repose, in which all the symptoms appear less severe. The cough lessens, the fever declines, the appetite improves, and a notable gain in flesh may ensue. Under such circumstances the patient, and physician also, may feel greatly encouraged ; but none of the physical signs indicating consolidation of the caseous area change their significance, and the symptoms of improvement prove delusive. Presently the process of softening begins (after some weeks, even many months), and with the softening, destruction of the pulmonary parenchyma and the formation of cavities. Caseous phthisis may come on in an apparently healthy individual—it may be in a robust subject, of a full habit. In a few months a marked decline in strength, flesh, and activity has occurred—all dating from the time of the acute cold (catarrhal pneumonia), since which the symptoms of pulmonary trouble have persisted.

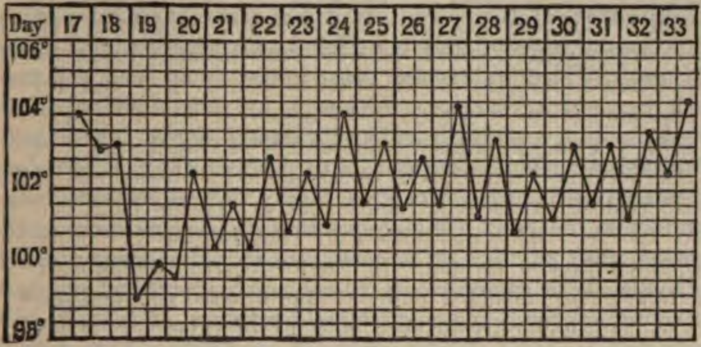

FIG. 38.—Temperature of Catarrhal Pneumonia becoming caseous.—Phthisis Florida.

In the acute variety, or *phthisis florida*, the whole course of the disease is run in a few weeks. It begins as a catarrhal pneumonia, involving almost the whole of one or parts of two lungs. It commences rather abruptly, with chilliness, fever, cough, pain in the chest, and rapid loss of strength. The temperature runs very high during the exacerbations, to 104°, 105° Fahr., or even higher, and there are considerable remissions and profuse and exhausting sweats. Owing to the sudden obstruction of so much of the breathing-space, there is marked dyspnœa. The cough is very troublesome, preventing sleep, and the expectoration is profuse, purulent in character, and often streaked with blood or bloody, but has not the rusty appearance of the sputa of croupous pneumonia. The body emaciates rapidly, the strength is soon utterly gone, and the appetite is entirely absent. The symptoms increase in intensity, so that in the course of a few weeks or months the case terminates in death. Rarely a remission in all the symptoms takes place, an improvement in the local and general condition follows, and thereafter the case pursues a more chronic form. In these cases of *phthisis florida*, a large part of one lung or parts of the two lungs are occupied with the catarrhal pneumonia, and the products of the inflammation undergo caseous degeneration, so that after death a lung may be a mass of cheesy deposit.

2. TUBERCULAR PHTHISIS.

Definition.—Tubercular phthisis is that form of pulmonary consumption characterized by the deposit of gray tubercle ; by the changes due to such deposit, its softening and extrusion, and the less or greater destruction of the proper tissue of the lungs consequent on these processes. Tubercular deposit in these cases, if not limited to, is chiefly in the lung, and the disease of the lung-tissue quite overshadows that of any other organ. Acute tuberculosis is a general deposit of the miliary tubercle, accompanied by symptoms of universal disturbance of the functions of the body. As it is a general and not a local disease, it is more appropriately considered with constitutional diseases.

Etiology.—That tubercular consumption is an inherited malady, is held by most authorities. Although, by some leaders of modern medical thought, a certain peculiar "vulnerability of constitution" is transmitted and not the disposition to phthisis, the fact is undoubted that, when tuberculosis exists in a family line, it appears from one generation to another. This disposition to consumption is closely associated with scrofula or struma. In early life struma manifests itself by glandular enlargements, a tendency to protracted suppuration, and the development, under irritative conditions, of tubercle. After puberty, the tendency of the strumous constitution is to tubercular deposit in the lungs. One of the factors in determining tuberculosis of the lungs is a badly formed thorax. The position at the apex, the favorite seat of tubercular deposit, may be due to the imperfect respiration at this point, owing to its position and conformation. All the

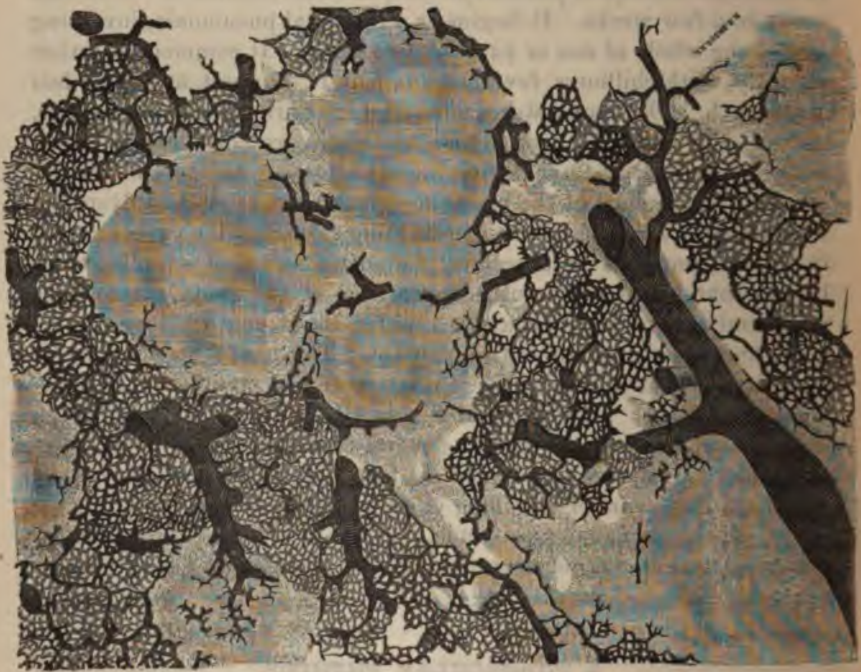

FIG. 34.—Miliary Tuberculosis. (Thierfelder.)

conditions which depress the bodily forces favor the growth and deposit of tubercle. Confined and foul air, excess of humidity, and rapid variations of temperature, are very influential elements in the sum of causes. Living and sleeping in badly ventilated apartments impair the quality of the blood, and invite disease to the lungs. A direct relation has been ascertained to exist between the amount of consumption in a given locality and the humidity of the air. Bowditch first ascertained this for Massachusetts, and the same fact was also shown in England. Variability of climate and rapid and extreme atmospherical vicissitudes have a most injurious effect on those having a tubercular diathesis. Elevation and dryness are as conspicuously beneficial as the opposite conditions are hurtful to those having a phthisical tendency.* The absence of sunlight, by contributing to anæmia, also favors the development of tuberculosis. Improper and insufficient food is an influential factor. The repugnance to fat, which is so often manifested by the phthisical, is unfortunate, since it is so necessary as a force-furnishing food. "Is phthisis communicable?" is a question much disputed, but which seems supported by many affirmative examples. The first experiments with the inoculation of tubercle, by Villemin, apparently proved its specificity, but subsequent researches rather diminished the confidence of those who adhered to this view, since it was shown that various kinds of animal and vegetable matter, especially when decomposing, produced the same results in certain animals when introduced under the skin or inserted into any of the tissues. It was soon maintained that it was the peculiar characteristic of the animal to produce tubercle on irritation, rather than the specificity of the tubercle-matter itself. Rabbits and Guinea-pigs are the animals especially which were found to possess the peculiarity that tuberculosis develops when any form of suppuration is induced in them. It came to be held, then, that certain individuals are, in respect to the development of tuberculosis, like these animals: when subjected to suppurative inflammation, especially in the glandular system, tuberculosis results. It is now maintained, however, that there is nothing specially characteristic in the histological structure of miliary tubercle. There are nodules of syphilis, of lupus, etc., that can not be distinguished in respect to minute structure from the nodules of tuberculosis; but the latter differ from the former in being specifically infectious. Thus, it has been clearly shown that general tuberculosis can be induced in animals by mixing tuberculous matter with the food eaten by them, and by causing them to inhale the sputa from tubercle-cavities. Probably the most conclusive proof of the infectious nature of true tubercle is afforded by the inoculation of the cornea with tubercle-matter, and the production of general tuberculosis, when no other kind of matter effects such results. This is, indeed, regarded by Cohnheim as the *experimentum crucis*—as proving beyond doubt the specific infective property of true tubercle.

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* See Lombard, "Traité de Climatologie Médicale," etc., tome iv, Paris, Baillière et fils, 1880, p. 404, *et seq.*

Such was the position of this subject when Koch's remarkable discovery was announced. By a special method of preparation and staining, he was able to demonstrate a specific bacillus—the *Bacillus tuberculosis*. This parasite has great vitality, and therefore easily resists ordinary destructive agencies. It is contained in the sputa, in the matter of cavities, in the giant-cells of tubercle, etc. The diagnosis of obscure cases may depend on the recognition of this organism. The number of bacilli is in direct ratio to the intensity of the destructive process, according to Balmer and Fraentzel (*Berliner klinische Wochenschrift*, No. 45, 1882). Hence, when the disease is slow or is stationary, the number of these bodies is small, and those present contain no spores. It appears, also, that if the bacilli are found in great numbers in the sputa, which is a favorite soil for their growth, they are less numerous in the walls of the cavities. If the fever is slight, the bacilli are imperfectly formed. Not only are they contained abundantly in the sputa, but also in the expired air of phthisical subjects. The presence of air is not necessary to their growth and development, for they exist in closed cavities of strumous and tubercular abscesses. The relation of these bacilli to tubercle is further shown in the fact that they have been found in various forms of tubercular ulceration, and in all parts, the seat of this process.

Although the bacillus tuberculosis has been widely accepted, opposition is developing in various quarters. Especially has this opposition to Koch's views taken form in Vienna, and hence something must be allowed to the jealousies which have existed between the two cities. Dr. Spina, who has long held the position of chief assistant to Stricker, may therefore be supposed to be fully acquainted with the technique of the processes. He opposes Koch at all points, and maintains that the bacillus has not a constant form, but varies with the tissue and local condition; that it is not essential to the tubercular process, and that it is frequently absent in undoubted tubercular disease. From the practical side, Koch's theory has received a severe blow in two cases which have recently occurred at Nothnagel's clinic. In both tuberculosis was diagnosticated, because the bacilli were detected in the sputa. Yet, on *post-mortem* examination, both were ascertained to be examples of bronchiectasis, and no tubercles existed at any point. It follows, from these facts, that the parasitic nature of tubercular phthisis must be regarded as *sub judice*. The attitude of the reflecting physician should be that of receptivity, but he should not rush to the conclusion that the parasitic nature of phthisis is proved.

Given a specific bacillus, the communicability of phthisis would thereby seem to be established. Before this discovery the evidence of its transmission, especially from husband to wife, had been accumulating. Indeed, a belief in the contagion has prevailed to a less or greater extent from the earliest times. In Southern Europe and in North Germany, especially during the last century, this belief was almost universal. In a treatise on consumption, by Dr. Morton, published in 1694, and in Dr. Young's work on the same malady, which appeared early in this century, this view was strongly maintained. In England, various writers have alluded to this subject, and by several of eminence the fact of the contagious nature of phthisis has been considered established by the clinical evidence. Thus, Dr. Walshe, who originally opposed the belief in its communicability, subsequently practically admitted it. In 1867 Dr. Budd published a paper in the "Lancet," in which he maintained that the disease is communicable, and that the tuberculous matter in the sputum contains the specific, infective material. Whether the bacillus of Koch continue or not to be regarded as the agent of infection, it must be admitted that strong reasons exist for adhering to the view of the contagious character of tuberculous phthisis. But, as every germ needs a special soil for its growth, so the specific infective material of consumption must fall into a properly prepared organism, to proceed to full development.

If the bacillus tuberculosis is to be found in all suppurative inflammations of tubercular origin, we have in this fact an explanation of the occurrence of pulmonary tuberculosis as secondary to catarrhal pneumonia, to pleuritis, to glandular affections in various situations, etc. The conditions of its spread from a point where it first appears, the manner of its propagation from one individual to another, and the descent by inheritance, are points of which we have no certain knowledge. Especially is it difficult to reconcile the fact of heredity with the present knowledge of the behavior of the parasite.

Pathological Anatomy.—The miliary tubercle is a grayish-white, translucent, and semi-solid granulation, about the size of a millet-seed, composed of a reticulum, with cells, giant-cells, and nuclei, the cells resembling white blood-corpuscles except that they are smaller, and the giant-cells having many nuclei. The reticulum is an extremely delicate network, inclosing the cells in its meshes, the giant-cells being placed nearly at the center of the granulation. The specific element is a minute, flat, long bacillus, rounded at its extremities, straight or curved, and beaded in appearance. Bacilli are usually single, but are sometimes found in pairs, and they multiply from spores, apparently only in the body, since they require a high temperature for their growth. They are found in the cells of tubercle nodules or masses, especially in the giant-cells. Tubercle, then, according to the bacilli theory, consists in the deposit and pullulation of the bacillus tuberculosis, and it is the matter thus formed which is deposited in the lungs, and constitutes pulmonary tuberculosis. According to Rindfleisch, tubercle takes its origin from the connective-tissue cells of the blood and lymph vessels, and the first deposits occur at the point where the bronchioles unite with the acini. (A group of acini communicating with a bronchus is a lobule.) A whitish nodule—a tubercle granulation—is thus formed around the termination of the bronchiole in the acini, in the angle at their point of junction, the deposit being in the connective tissue. The nutrient vessels are included in the granulation, and their adventitia become swollen and infiltrated. It is this development of tubercle in the connective-tissue cells of the adventitia that weakens the vessel, and which may finally cause a rupture and hæmorrhage. So many vessels at the apex are occluded by the mass of the deposits, that the pressure in the remaining vessels is much increased. When the walls of the vessels are infiltrated, rupture occurs the earlier by reason of the increased pressure from the cause just named. Tubercular deposition also takes place abundantly in the bronchioles, not only those in immediate relation to the lobules, but for some distance beyond. The lymphatics distributed to the mucous membrane are infiltrated, and next those of the peribronchial space, so that all around the alveoli and bronchioles are thickly placed masses of tubercle granulations. The intervening connective tissue is also densely infiltrated. With the deposit of tubercle, there are associated the results of inflammation excited by the presence of these granulations. According to Rindfleisch, a desquamative pneumonia plays an important part in the subsequent changes. The cheesy transformation of the products of catarrhal pneumonia, atelectasis, bronchial dilatation, assist materially in enlarging the area of structural changes. The masses of miliary tubercle, in a variable period after their deposition, and often within a few weeks, undergo a cheesy transformation, by which they are brought into close resemblance to the cheesy products of caseous pnuemonia. It is a process of fatty degeneration, beginning in the central portion of each nodule. In acute tuberculosis, to be studied hereafter, the gray granulation is disseminated throughout both lungs. In the pulmonary tuberculosis, the deposits occur chiefly in the superior lobes, and are often limited to the apex, but are very rarely indeed confined to one lung, and, when this is the case, the left is more often attacked than the right. When the process of cheesy transformation is completed, the resulting mass is opaque, yellowish, and has the friability of cheese. The infiltration of all the parts, ultimately, of which the parenchyma of the lungs is composed, the closure of the vessels and entire arrest of the nutritive supply, and the compression exerted by the contracting connective tissue, necessarily cause a necrosis of the pulmonary elements. When the stage of softening comes on, the products, although having a puriform appearance, are not purulent. Inflammation and suppuration are excited in the tissues, with the necessary result of disintegration. On the surface of the mucous membrane the destruction of the tissue in and about the site of the tubercle granulations is an ulceration; in the mass of disease in the body of the lung the destruction of tissue produces a cavity. The fluid matter resulting from the softening of the yellow tubercle is homogeneous, of the consistence of cream, and having a greenish-yellow or grayish color. Mixed with it are necrosed pulmonary elements, solid particles of a yellowish color, and the whole is contained in a small cavity, surrounded by masses of cheesy tubercle. The softening proceeds from the center to the periphery, and in its progress the pulmonary elements are disintegrated with it. When discharge of a cavern takes place by the ulceration opening a bronchus, or, according to Rindfleisch, by the tubercular ulceration of a bronchus, the elastic fibrous tissue may be recognized in the sputa. Large caverns are formed by the breaking down of the intervening septa and the coalescence of smaller ones. The increase in the area of destructive ulceration is greatly promoted by the attacks of catarrhal (desquamative) pneumonia, which induce softening and dilatation of the bronchi, and collapse of lobules (atelectasis); and catarrhal products fill the alveoli and bronchi, and there caseate. Cavities are produced under these circumstances by the softening and extrusion of the caseous masses as described under the head of caseous phthisis. In this case the tubercle granulation is the exciting cause of the catarrhal pneumonia; in the former the products of catarrhal pneumonia undergo the caseous change in consequence of a peculiar "invulnerability" of the constitution, without which the catarrhal products would pass through the ordinary changes. Dilatation of the bronchi, or bronchiectasis, plays an important part. In catarrhal pneumonia, the walls of the bronchi yield in consequence of an extension of the inflammatory process to them, and, as the existence of dyspnœa renders greater inspiratory efforts necessary, and as the area for the admission of air is much reduced, obviously the interbronchial pressure is raised, so that greater force is exerted against the weakened tubes. According to Rindfleisch, the walls of some cavities are in part formed by dilated bronchi. Cavities, still extending, have no proper boundary, and are surrounded by tubercle and caseous masses undergoing softening, and by detritus of the lung-tissue. Others are lined by a connective-tissue membrane, which continuously pours out a puriform matter of a greenish-yellow, often having a foul odor by reason of decomposition from the presence of air. When the cavity is recently formed, not only are its sides ragged and uneven, but large bands traverse it, remains of pulmonary tissue not destroyed. Other organs besides the lungs are affected. The *pleura* is usually the seat of a chronic inflammation; it may take the form of a dry pleurisy, and close adhesions form universally, so that the cavity is obliterated ; or the adhesions may be local and partial when they are chiefly at the apex ; or a neo-membrane is formed, and both the pleura and the new membrane may become tuberculous. Extensive effusion may be formed in consequence of the rupture of a cavity and the escape of its contents, when a pyopneumothorax results. A cavity perforated and firm adhesions having formed, the pleura may ulcerate and discharge take place through the thoracic parietes, a fistula remaining. The *bronchial glands* enlarge by hyperplasia of their contents, which undergo caseation. They may be dry and cheesy, or suppurate and discharge, the pus finding an exit by the trachea, or by a bronchus, or by the œsophagus. In infants and children, enlarged bronchial glands may compress the trachea or bronchi, or the pneumogastric, and thus give rise to suffocative attacks. It may be well to mention that the late Dr. Fuller, of London, had secondary pyæmic abscesses of the brain, from suppurating bronchial glands. The *larynx* always suffers from some morbid change in pulmonary tuberculosis. From simple hyperæmia up to extensive tubercular ulcerations, destroying the epiglottis, vocal cords, etc., there are numerous gradations in the severity of the lesions. Tubercular ulcerations also occur in the œsophagus, stomach, and intestines, but the point of greatest development of the ulceration is the lower part of the ilium and the large intestine. The tubercular troubles of the intestinal canal are found in two stages : the initial deposit, and the softening and destruction of tissue or ulceration. The peritoneum is granulated, and chronic lesions of the peritoneum coincide with the formation of ulcers in the intestine. The liver is usually in an advanced stage of fatty degeneration, but in rare instances the change is that of amyloid disease. In the kidney, the amyloid degeneration is more common than the fatty. Tubercular ulcerations are often found all along the urinary tract.

Symptoms.—There is a peculiar type of constitution, as a rule, associated with tuberculous phthisis, which, being present, may serve to excite suspicions, at least, in obscure and doubtful cases. These peculiarities are observed in growing youths and young men, and may be described as follows : They are tall and rather thin ; the neck is long and small ; the thorax flat, narrow, and having but little expansile mobility ; the muscles, especially of the chest and neck, are thin and poorly developed ; the intercostal spaces are wide ; the hair is fine, the eyelashes long ; the eyes are large and bright, the sclerotic glistening ; the skin is transparent and thin, the color quickly changes, and the veins are blue and distinct ; the fingers are long and tapering, but their extremities are incurved or club-shaped. These subjects possess certain moral and mental characteristics also : they are impressionable, the disposition is variable ; they are fond of activity, but fatigue easily ; others are more phlegmatic, speak slowly, and differ in complexion, being dark, with thick, muddy skins. When these peculiarities of constitution coexist, with an hereditary tendency to phthisis, they possess a high degree of significance. In such subjects, cough, losing flesh and strength, with a red line along the margin of the gum, are strongly indicative of the onset of phthisis, even when the physical signs may not be positive. A large proportion of the cases begin by loss of appetite, indigestion, decline in weight, without cough or any symptom referable to the lung. In women these symptoms are accompanied by disorders of menstruation. Again, an attack of hæmoptysis may be the first symptom. Most usually, the onset of the disease is characterized by a short, dry cough, which is rather more troublesome at night, preventing sleep, some shortness of breath, pains in the chest, either wandering or fixed in the position of an intercostal nerve, or a sharp stitch indicative of pleurisy, some nocturnal perspiration, confined at first to the neck and face, decline in flesh and strength, poor appetite, and often, more or less diarrhœa. At this period, too, some alteration of the voice is beginning to be perceptible and bronchial hæmorrhage occurs. The progress of the case is more rapid if the fever now appears. This may be an early symptom; it may be postponed until the period of softening. The action of the heart is excitable and is accelerated by slight causes from the very beginning, and the pulse is soft and compressible, the tension of the vessels being low. The usual type of fever in the beginning is the quotidian. There is a daily morning remission, an evening exacerbation terminating in a sweat—the co-called *hectic fever* (*septicæmic fever*). The type may be double quotidian—two paroxysms of fever each day—the first in the morning, the second at night. The range of temperature at this period is not great, the minima about 98° Fahr., the maxima 102° Fahr. The range of fever-heat is an important indication of the degree in which the morbid processes are proceeding, especially those involving the lungs. In illustration of this may be mentioned *phthisis florida*, in which the highest temperature of this disease is attained because of the immense extent of the caseous deposits undergoing softening and extrusion. As the case proceeds, all the rational signs become aggravated. The appetite is almost gone; in severe paroxysms of coughing, in the last straining effort to dislodge the sputa, vomiting is excited, an accident very apt to occur after meals. The diarrhœa also increases, and becomes very difficult to restrain. The cough, also, grows more troublesome and painful, the expectoration more abundant, and the voice harsh and husky. Difficulty of swallowing comes on in consequence of ulceration of the epiglottis, and sometimes the attempts at swallowing are embarrassed by the dropping of particles of food and drink into the glottis, exciting violent suffocative attacks. The expectoration assumes a different character at various periods. At first there is brought up, often with a great deal of effort, some frothy mucus; after a time the sputa become purulent or muco-purulent, greenish or greenish-yellow in color, without air, and without viscidity, unless there is a complication of pneumonia, when the sputa will have a grayish, vitreous, adhesive character, and may also present a slightly rusty aspect from the admixture of blood, or may be simply streaked with blood. These adhesive sputa may be seen in large muco-pus expectorations, as isolated particles. The sputa often have a striated appearance, at one time supposed to have much significance, but now known to be produced by the diminution of the cellular elements and the presence of deformed and atrophied cells and of granules—changes of a degenerative kind due simply to retention in the lung. A very significant element in the sputa is the presence of elastic fibers of the pulmonary tissue. These bodies are most easily detected by boiling the sputa in a solution of caustic soda in distilled water (18—100) according to the method of Fenwick. The most distinctive element is the *bacillus tuberculosis*, which is necessary to constitute tubercle, according to the modern conception. Well-developed specimens can usually be obtained from the expectorated matters, even in cases of the so-called "miner's lung." The next change in the sputa is the characteristic impressed on them by formation in small cavities. They then consist of two parts, a frothy muco-pus from the bronchi, and isolated, globular, compact masses without air, of a greenish or grayish color; when allowed to stand, the former rises and the latter sinks to the bottom, and, if put in water, sinks quickly. The quantity of expectoration varies; in the beginning, because then it is derived from a bronchial catarrh; afterward, according to the extent of the cheesy masses undergoing softening, the size of the resulting cavities, and the degree in which bronchiectasis exists. When there is a large cavity, quantities of little more than pus are expectorated. When the patient lies in a position to permit accumulation to take place, the expectoration may be suspended, but, when the position is changed, the pus is discharged in a stream. Sputa streaked with blood and rusty sputa have already been alluded to; but expectoration of blood, or hæmoptysis, is a different affair. According to some, phthisis may be due to pulmonary hæmorrhage. This notion arose from the clinical fact that hæmoptysis is sometimes the first symptom of the disease, and after its occurrence there is an immediate development of the symptoms. The presence of blood-clot is supposed to excite an irritation which has for its ultimate effect the formation of tubercle. The most generally accepted view is, that hæmorrhage is merely a symptom, and a symptom that may occur at any

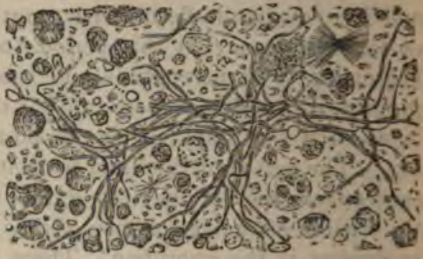

FIG. 85.—Fragment of Lung-Tissue and Sputa. (Beale.)

period. If we accept Rindfleisch's demonstration, that the formation of tubercle begins in the connective-tissue cells of the adventitia of the vessels, there can be no difficulty in comprehending the early appearance of hæmorrhage in the course of phthisis. At any subsequent period, the extension of the area of tubercle formation may be accompanied by hæmorrhage. Again, hæmorrhage, often considerable, may be due to the erosion of an unclosed vessel in the process of destruction, ending in the formation of a cavity. The amount of blood lost varies from a drachm or two to several pounds. The blood is bright colored, more or less aërated, and comes up with coughing; but a sudden large hæmorrhage may pour up in a stream and be ejected by the nose as well as the mouth. A considerable part of the blood may be swallowed, and subsequently vomited, and, as it is then acted on by the gastric juice, presents the appearance of hæmatemesis; but the history of the case, the rational and physical signs of pulmonary disease and the absence of stomachal disease will afford the data for a correct diagnosis. After the hæmorrhage has taken place, and the flow is arrested, for some days clots of small size and blackish in color are expectorated. Occasionally there are indications of the approach of a hæmorrhage, the significance of which the sufferers from them soon learn: these are a feeling of warmth in the chest, oppression of breathing, excited action of the heart, and a rather sweetish and saltish taste in the mouth. Usually, nothing in the nature of a warning of the approaching hæmorrhage is observed. When the blood-taste is experienced, the mouth should be examined, for the gums may be the source of the hæmorrhage. Bleeding from the posterior nares may also be confusing, as there may be a coincident cough. A pulmonary hæmorrhage may be vicarious of the menstrual flow, and it may be determined by the sudden arrest of hæmorrhoidal bleeding.

3. FIBROID PHTHISIS.

Definition.—By this term is intended a form of consumption characterized by hyperplasia of the connective tissue of the lung and atrophy and degeneration of its proper structure. In this respect the disease corresponds to fibroid liver, fibroid kidney, etc., but the changes do not begin in and are not limited to the connective tissue. Bronchial inflammation, bronchiectasis, bronchorrhœa, and exudative pleuritis are among the initial changes, the pulmonary tissue being involved subsequently. Ultimately tubercular deposits occur, and the lesions produced by these are added to those already existing in the connective tissue and the bronchi.

Etiology.—Heredity is concerned to the extent that the type of pulmonary tissue favorable to the development of this disease is transmitted. It is a disease of mature life, after the middle period, and is extremely rare before thirty. Next to heredity, chronic bronchitis and a form of pleuritis characterized by extensive organized exudation are the most influential factors. The causes of chronic bronchitis are, therefore, indirectly the causes of fibroid phthisis.

Pathological Anatomy.—The mucous membrane of the bronchi is of a dark red in the more recently inflamed parts, of a slate-color in the older, traversed by dilated vessels, its glands much thickened and elevated above the general surface. The sub-mucous connective tissue is thickened, the muscular layer hypertrophied at first, but in the further progress of the case the whole tube is softened and dilated. These dilatations may be fusiform or sacculated. The latter predominate, and are often mistaken for cavities, the resemblance being the more striking if the dilatation contains an accumulation of pus. The atrophic changes in the walls of the bronchi are not the only factors concerned in producing dilatation. The force of the expiration in coughing, the contraction of the adjacent connective tissue, and of pleural adhesions, are also concerned. From the bronchi the inflammation slowly extends to the peribronchial, perivascular, and interlobular connective tissue. A hyperplasia of its constituent elements takes place, with the result to compress the vessels, the acini, and the bronchioles. The contraction of the newly formed connective tissue, by cutting off the blood-supply and encroaching on the neighboring parts of the pulmonary tissue, causes an atrophy. Some of the lobules collapse (atelectasis); all within the affected area contain less blood, and are narrowed by pressure. The collapsed lobules undergo the changes already described. In the progress of these cases catarrhal pneumonia ultimately plays a part; the bacillus tuberculosis appears in the caseous masses, and these undergoing softening and extrusion, cavities are formed. So that the cases of fibroid phthisis, although differing in their rate of progress and in the greater importance of the sclerosis to the other morbid processes, nevertheless are brought into close relation to the other forms of phthisis. A considerable increase of the connective tissue of the lungs occurs in chronic tubercular phthisis; the longer the duration of the disease, in fact, the greater is the development attained by it. The walls of the cavities are composed of a dense layer of connective tissue, closely united to the same tissue of the lung. In caseous pneumonia there is less production of connective tissue, because of the rapid progress. In a fibroid lung the cavities do not attain to great dimensions; they appear as interspaces in the dense trabeculæ. When these intervening portions of the condensed tissue are divided, they are ascertained to be exceedingly firm, of a grayish or slate color, containing here and there patches of brown pigment, and possess but little vascularity. The early compression and closure of the vessels is a source of mischief to the heart. The pulmonary circulation being obstructed over a considerable portion of the lung, the right cavities yield to the increasing pressure and dilate. There is, therefore, a stasis of the venous circulation; the liver enlarges, and ascites is produced; the kidneys are congested, and albumen is present in the urine. These complications develop toward the close of the malady.

A considerable proportion of the cases of fibroid phthisis originate in "dry pleurisy"—characterized by the formation of extensive solid exudation. Three varieties of this form of pleuritis are recognized: the fibrinous, the croupous, the proliferative; * but it is probable the three processes are mixed in every case. An extension of the pleuritis takes place by contiguity of structure; the connective tissue of the lung is entered by the interlobular fissures; the peribronchial and perivascular connective tissue is invaded and undergoes proliferation, and then the alveoli become affected. Thus the morbid process comes to involve all the pulmonary elements. At first a general congestion of the affected parts ensues; then the exudative process occurs, and at length the deposit and pullulation of the bacillus are manifested by caseation, ulceration, and extrusion of the products of its development. The general course of the structural alterations of the pulmonary parenchyma is as already described.

Symptoms.—Fibroid phthisis is the most chronic form of the disease; its early history is that of bronchial catarrh, or of dry pleurisy of the exudative form; and it is not until after months, even years, that, extension taking place to the lungs, the progress becomes more rapid. For months there is merely a dry cough, not very troublesome, but persistent. The expectoration is slight, and is nothing but mucus. The appetite is but little impaired, and the weight and strength are not materially reduced. During the fall, winter, and spring months the symptoms increase in severity; the cough becomes more troublesome, and the expectoration more abundant and having the appearance of muco-pus. The symptoms ameliorate during the warm months, but to increase again with the changeable weather of winter. After two or three years of this alternation, there is less and less improvement in the warm months, but the symptoms of catarrh continue throughout the year. Fever comes on toward evening, the temperature at first rising to 100° Fahr. The appetite lessens, digestion becomes poor, and the body-weight progressively declines. The cough is harassing and prevents sleep; the expectoration becomes more profuse and entirely purulent; and the food now and then comes up in the attempt to clear the larynx and fauces. Some difficulty of breathing is experienced; the pulse is small and weak; the skin is warm toward evening, while slight chilliness is felt in the morning, and sweating occurs during the night. As the disease advances, the temperature reaches 101° and 102° in the evening, but it does not attain to the altitude reached in caseous or tubercular phthisis. When the bronchi dilate, the expectoration becomes profuse, especially in the morning—

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* Sir Andrew Clark's "Lumleian Lectures," "Lancet," vol. i, 1885.

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a cupful or more may be brought up in an hour or two. Fragments of fibrous tissue only appear in it when cavities are forming. At this period there may be one or more hæmorrhages. Detritus of caseous matter containing the bacillus, softening, is found in the sputa only at this later period. The onset of tuberculosis is announced by increase of dyspnœa, rise of the temperature, alterations in the voice, and diarrhœa. The development of the connective tissue and the compression of the vessels lead to dilatation of the right cavities of the heart, stasis of the venous system, and congestion of the liver and kidneys. Œdema of the feet and ankles is first observed; then swelling of the legs and scrotum, and ascites appear.

Physical Signs of Phthisis.—There are no points of difference as respects the physical signs of phthisis; hence the three forms may be considered together.

The abnormality in the development of the chest, which is observed in phthisical subjects, has been already described. In the movements of the ribs during expansion in *inspiration*, deficiency may be observed to exist on the diseased side. On *palpation*, increase of the vocal fremitus exists over consolidated lung and over cavities, and is diminished or wanting over effusion in the pleural cavity. The percussion-note has great variety. All shades of dullness exist. If the consolidation is not complete and some air still enters the diseased area, the note is high-pitched, but with a somewhat tympanitic quality; but if the tissue is entirely without air, then the note is high-pitched and hard in quality. The change in sonority may be unilateral or double, but if double

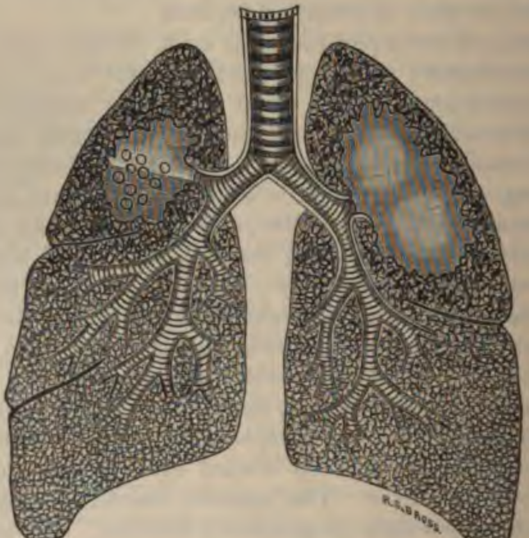

FIG. 86.—Cavities; one partly filled, one empty. (Da Costa.)

it is not necessarily symmetrical; it may be infra-clavicular on one side, infra-spinous on the other. The dullness may be due to various causes —to a pleuritic effusion, to pneumonic consolidation, or to a tumor or cyst. The extension of the area of dullness and the increase in hardness or the disappearance of the tympanitic quality may indicate the increase of the tubercular or caseous deposition. The change in the sonority of the lung is most usually at the apex, but it may be in any situation. During the process of softening and extrusion there is no change in the character of the percussion-note until excavations have formed; even then there will be no change, unless the cavity be large and near the surface. The percussion-note may present a nearly normal sonority or it may be exaggerated over a cavity; it may have a metallic clang, or amphoric quality; it may, if the cavity communicate with a bronchus, have the cracked-pot sound (*bruit de pot fêlé*). The last is produced by strong percussion, the vibrations occurring in the walls of the cavity and in the column of air in the bronchus, A cavity in which pus has accumulated may furnish a dull sound; when emptied, the amphoric sound will return. On auscultation the sounds audible will present great variety. The vesicular murmur will be unimpaired in those parts free from disease; it will be feeble or indistinct if many bronchioles are obstructed; it will be rude or blowing if the bronchioles are narrowed; inspiration will be jerking and expiration prolonged and blowing if the lung has lost its elasticity from any cause. These signs are much less significant when they occur on the right than when they occur on the left side (infra-clavicular regions); in the former situation, they are, so to speak, normal. Next to these modifications in the respiratory murmurs are certain adventitious sounds, or *râles*. The earliest of these audible in the infra-clavicular region usually is a fine, dry, crackling sound (sub-crepitant) appearing at the end of inspiration, and sometimes requiring a deep and full inspiration to develop it. This *râle* may be temporary, when it has but little significance. The extension of the inflammation to the larger bronchi induces more abundant secretions, and the sub-crepitant *râle* becomes a distinctly moist sound, and audible over a larger area, and coarser sounds also moist—mucous *râles*—are mixed with them. With these *râles* changes in the respiratory sounds take place: inspiration has a distinct *blowing* character which approximates to and ultimately does become *bronchophonic*—i. e., the sound of the movement of the air in the bronchial tubes and of the voice are communicated to the ear directly, the solidified lung acting as a good conductor, the respiratory or vesicular murmur having disappeared. These are the sounds of consolidation, and of softening up to extrusion. When cavities form, new sounds become audible, but it is not always easy to differentiate between bronchophony and *amphoric* and *cavernous blowing*, the signs of a cavity. Amphoric blowing and amphoric voice are signs of a cavity, if correctly interpreted ; the *cavernous sounds* produced in a large cavity with thin walls are more significant. To these must be added *metallic tinkling*, which is heard in perfection in hydropneumothorax and under similar conditions when the cavity is large.

Course, Duration, and Termination.—The course of phthisis is much influenced by its form. Phthisis florida, or acute caseous phthisis, runs its course in a few months, and not often with intermissions, although it does sometimes intermit, and then pursue a more chronic course. Its usual course is continuous—a large part of one or of both lungs may be occluded, softening occurs, and high fever with rapid emaciation soon exhausts the powers of life. The usual type of caseous phthisis is chronic ; there are repeated bronchial attacks and gradually increasing consolidation, the interval between the attacks being characterized by varying degrees of improvement, but with a general tendency toward decline. In many, it is true, under judicious management, the catarrhal process is arrested, absorption of the caseous matter takes place in part, the rest is extruded, with more or less destruction of tissue ; cicatricial tissue supplies the place, contraction ensues, with subsequent retraction of the chest-wall, and thus, in a limited sense, a cure is effected. In other cases the course is less marked by intermissions, the caseous deposits are extensive, and there are hæmorrhages, fever, emaciation—the symptoms continuing until death. While the duration of the former type may be two, three, and as much as five years, or during the ordinary duration of life, the latter do not often extend two years. The tuberculous form also pursues two different courses : one chronic, developing slowly, lasting two years or more ; the other more rapid, the whole course being terminated within a year. The degree in which broncho-pneumonia, atelectasis, and dilatation of the bronchioles occur, the extension of the tuberculosis to the larnyx and intestinal canal, and the number and severity of the hæmorrhages, are important factors in bringing about a fatal result. So long as the tubercular deposit is limited to the lung, is slight in extent, there is a possibility of recovery by extrusion, shrinking of the lung, and retraction of the ribs. The most chronic of all the forms of phthisis is the fibroid. The course of this may occupy several years, indeed an ordinary lifetime, and prove fatal at last. Of all the forms, it offers the best prospect of a cure, if the changes are not too extensive. The initial period, terminating in a bronchiectasis, may occupy a number of years ; at first, for several years, there is winter cough only, the warm season being free, or nearly so ; when the connective tissue of the lung is invaded the progress is more rapid, for then atelectasis and caseation enter as elements into the destructive changes. Finally, tuberculosis is ingrafted into the morbid process, which then advances more rapidly, because not only the lungs, but the larynx and intestinal canal, become diseased; the range of temperature rises higher, and emaciation proceeds at an accelerated pace. Phthisis is the great enemy of the human race, since nearly two sevenths of the deaths from all causes are due to this disease. But a few years ago, a cure of any case was regarded as hopeless; but within recent times the improvements in our knowledge of the local conditions and in the means of treatment have led to better results, and cures are now not uncommon.

Diagnosis.—The diagnosis of phthisis can not be doubtful after the initial period. Incipient phthisis may be confounded with atonic dyspepsia. A cough may be present in atonic dyspepsia—the so-called stomach-cough. The natural differences in the sonority and the respiration of the right and left infra-clavicular regions may materially contribute to the error. Attention to this, and to the fact that there is no point of irritation about the air-passages to account for the existence of a cough, will settle the doubts. More frequently, in malarious regions, is hectic fever confounded with intermittent, since in the latter there is usually some cough. This mistake is made when the pulmonary disease is quite advanced, so that the error is either from ignorance or carelessness. In phthisis, independently of the physical signs, the fever has been preceded by a period of cough, and loss of flesh and strength, whereas in intermittent these symptoms have followed the access of fever; in phthisis there is not, in intermittent there is, an enlarged spleen; in phthisis the hectic is not arrested by large doses of quinine; in intermittent the fever is arrested and convalescence is at once established. A careful study of the physical signs ought at once decide the question. Laryngeal symptoms are often so pronounced in the beginning as to obscure the pulmonary affection. Indeed, the disease in the lungs is referred by some to the larynx, to which it is regarded as strictly secondary. This error has arisen from the fact that considerable infiltration of the lung may exist without seriously impairing its sonority, or changing or modifying the vesicular murmur. When tubercular deposits occur in the larynx, the tone and quality of the voice are quickly affected, so that the latter may seem to be the only seat of tubercular deposit. Although, to determine this question, time may be necessary, the coexistence of pulmonary disease ought to be suspected, because of the relation known to obtain between them. The most important diagnostic question relates to the difference between caseous and tuberculous phthisis. The sections devoted to these two forms have indicated the clinical and pathological differences; nevertheless, it will be useful to state briefly the points which serve to distinguish them. Tubercular phthisis is distinctly hereditary; caseous phthisis is not hereditary, but occurs in the scrofulous. Tubercular phthisis occurs at all ages; caseous, from youth to middle age. Tubercular phthisis occurs insidiously with catarrh of the bronchi and larynx; caseous results from acute inflammations of the bronchi and lungs. Tubercular phthisis is more often than the caseous a cause of pulmonary hæmorrhage. In tubercular phthisis the lesions are apt to be on both sides; in caseous, on one side. In tuberculosis of the lung, tubercle may be widely disseminated without any striking physical signs; in caseous phthisis the caseous deposits produce very pronounced physical symptoms. The laryngeal symptoms are much more common in tubercular than in caseous phthisis. The progress in tuberculous phthisis is more rapid and the mortality greater than in caseous. Fibroid phthisis is distinguished from the other forms by its slow progress, by the long period of bronchial troubles before the pulmonary lesions begin, by the merely purulent expectoration, without fibrous tissue, until late in the progress of the case, and by bronchial dilatation long before the cavities by excavation form.

Treatment.—When a phthisical tendency exists, prophylaxis becomes highly important. Although not often consulted, physicians should discourage, directly and indirectly, the marriage of the phthisical. Children inheriting the dyscrasia should have a careful physical training, substantial food, warm clothing, and exercise in the open air without exposure. They should be guarded against attacks of bronchial catarrh, of measles, and whooping-cough, for in these diseases the seeds are sown of future mischief. As humidity is such an important factor in the etiology of phthisis, and as dryness and elevation are climatic conditions of the greatest utility, if possible, the growing child should be separated from the one and placed in the other. Singing should be encouraged, since that tends directly to improve the nutrition of the lung, especially of the apex. Cold bathing should be practiced every morning to diminish the susceptibility to cold. Catarrhal attacks occurring should receive prompt attention, and any lingering remnant of local morbid action should be carefully removed. The tendency to such attacks and the removal of the effects produced by them are equally controlled by the iodides (iodide of iron) and cod-liver oil. As phthisis is preëminently a wasting disease, it is highly important to put the organs concerned in nutrition into the highest state of efficiency. In tubercular and fibroid phthisis, among the earliest symptoms are stomach disorders, poor appetite, atonic or acid indigestion, and especially repugnance to the fatty elements of food. The mineral acids, with a bitter, such as tincture of nux vomica, are especially serviceable. If there be acid eructations, pyrosis, and heartburn, the mineral acids, especially nitric (ten to fifteen drops, well diluted, *ter in die*), should be administered before meals; but, if the condition be atonic indigestion, the acid should be given after meals. The nux-vomica tincture should be given before meals—fifteen drops in water. The aliment should consist of easily digested articles of diet, and the stomach should not be overloaded under any circumstances. It should never be forgotten that it is not the quantity swallowed, but digested and assimilated, which contributes to the nourishment of the body. There are certain tonics to the stomach which stimulate the organ to more efficient work, that are very beneficial in promoting the nutrition of the body. These are, besides the bitters and mineral acids mentioned above, small doses of arsenic and silver, and alcohol. Arsenic is deserving of special commendation—in incipient phthisis, to promote the appetite and favor tissue-forming, while it corrects the disordered state of the stomach mucous membrane, and as a remedy for chronic tuberculosis and fibroid lung. The author must impress on his readers that arsenic must be given in small doses, as it is to be continued for a long period (two drops three times a day). The oxide of silver performs much the same office, but its administration must be brief, because of the danger of coloring the skin (Argyria). Small doses of alcohol after meals (half an ounce for adults) are highly useful to promote appetite and tissue-formation. Physicians should not encourage the dangerous notion that whisky is antidotal to phthisis. Fibroid phthisis appears to be produced by chronic alcoholism. Large quantities of alcoholic fluids impair the function of digestion, and lessen tissue-forming; hence the amount named—certainly not more than twice as much—should not be exceeded. The utility of cod-liver oil in incipient phthisis is very great. As the power to digest fats is confined within narrow limits, and as the ability to dispose of them is relatively less in consumption, the dose of cod-liver oil should be prescribed accordingly, from a tea- to a tablespoonful—a teaspoonful the usual dose. All in excess of the capacity to digest passes unchanged, and may be seen floating on the evacuations. The utility of cod-liver oil consists in the fact that it is a fat, having a special digestibility, owing to its containing bile elements, and is therefore peculiarly fitted to form the "molecular basis of the chyle." It is not useful in cases of *phthisis florida*, or in caseous phthisis characterized by large deposits, high fever, and diarrhœa; but in chronic tuberculosis and fibroid phthisis. In what form soever it may be given, it is better to prescribe it with a little ether (♏ xx—ʒ j), because of the action of the ether in promoting the flow of pancreatic fluid—a fact demonstrated by Bernard, and confirmed by clinical observation. Cod-liver oil may be given in the form of emulsion with the lactophosphate of lime, the compound hypophosphites, and the compound phosphates. The simultaneous administration of these remedies is good practice, and the emulsion may be allowed, if the quality of the cod-liver oil is good, but it should not be overlooked that an inferior oil may be disguised in an emulsion of this kind. The lactophosphate of lime, if well prepared, is a most valuable agent in the treatment of incipient and the more chronic cases of phthisis. The hypophosphites, although not deserving the encomiums first pronounced on them as remedies for consumption, are valuable agents to promote the constructive metamorphosis. It is doubtful whether the hypophosphites present any advantages over the phosphates, because of their chemical instability and rapid conversion into the phosphates. The lactophosphate of lime has the special advantage that it is a soluble combination of an agent very important to the construction of tissue. The last-named remedy may be given in a dose of a tea- to a dessertspoonful of the sirup three times a day, after meals.

Recent experiences have shown that creosote is an important remedy. There is reason to believe that it has distinct curative power in suitable cases. The more chronic forms, and the condition of slow but extending disease, there being but slight fever and the perspiration moderate, are those most amenable to the treatment by creosote. Beginning with one minim, the dose is increased by minims until the full capacity of the patient is reached. There are some remarkable instances of improvement by the accidental administration of large quantities—almost toxic doses. Although from one to five minims may be stated as the usual dose, much more may be given by judicious and gradual increase in the amount. It is wood creosote and not carbolic acid that is meant here; although there is very close correspondence, there is not identity of composition. Besides the stomachal administration of creosote, it can be given by inhalation vaporized. Guttman has shown that tubercle bacilli are destroyed by creosote in a $\frac{1}{4000}$ solution. Sommerbrodt finds that if a sufficient amount, approximating the standard of Guttman, is given, the result is most satisfactory. The amount required is estimated as one gramme for a man weighing 160 pounds. He begins with nearly one grain to a capsule, of which he gives three the first day, four the second, five the third, and on at this rate till twenty are taken daily, and the results have been highly satisfactory.

Large doses of tannic acid (℈j), increased to an amount that seems almost incredible, have also lately been used, it is said, with curative results.

As the presence of a parasite—the bacillus tuberculosis—has an intimate relation to the morbid process, it is important to check its development by aërial or volatile remedies that can be given by inhalation. The value of antiseptics by the method of *protracted inhalation* is just being recognized, but I urge on my readers that the utility of mode of inhalation should have their serious attention. The method consists in charging the air of a suitable apartment with a volatile remedy which the patient can breathe—for a considerable length of time—for a half-hour, an hour, or even longer.

The method of germicide treatment by rectal injection of sulphhydrogen and carbonic acid was brought forward with such loud promises of the good done by it that in an incredibly short time it was being practiced in all civilized countries. It fell into disuse in this country as rapidly as it was taken up, partly because the mode of operating is unpleasant, and the results, although undeniably good, are not sufficient to counterbalance the disgust. The method, however, proved that the inhalation of certain gases having germicide power is one of the most effective means of treating phthisis.

The gases and vapors now employed in this disease are chiefly carbonic acid, sulphurous acid, and sulphuretted hydrogen; but the vapors are more numerous. By the atomization of solutions the number and variety of medicaments employed in the treatment of pulmonary affections become enormous. I hope I may not be considered egotistic and prejudiced when I say that this mode of using the remedies is not good—is not effective, certainly, in anything like the proportion that the promoters maintain. I advise the inhalation by the protracted method of ethyl iodide, carbolic acid, carbolic acid and tincture of iodine, bromine (very cautiously) iodol, iodoform, oxygen, etc.

The time when the most good is accomplished by germicide agents is before the bacillus has penetrated so far that excavations are forming. There is more to be expected in the chronic cases without cavities, for here the element of time is in favor of the result.

Cases of phthisis have been recently related as cured by the inhalation of highly heated air. If the air is deprived of its moisture the temperature at which it can be inhaled ranges from 150° Fahr., to 300° Fahr. This method is based on the power of heat to inhibit or destroy the bacillus. It is probable that this mode of treatment will develop into a highly successful one.

Some of the chief symptoms require remedies to restrain them within proper limits, as cough, fever, sweats, hæmorrhage, laryngeal symptoms, and diarrhœa. These we consider in turn. If *cough* is very distressing, some relief becomes necessary, and the constant temptation is to resort to anodynes. Gargling the throat with a solution of bromide of potassium, applying a mixture of chloral and camphor by means of a camel's-hair brush to the fauces, the atomization of a solution of cocaine, or of morphine, are expedients temporarily beneficial. Fothergill's prescription of hydrobromic acid (diluted) and spirit of chloroform sometimes acts well, but is often inefficient. Of the principles contained in opium, codeine is the least objectionable; it causes less disturbance of the digestive organs, and has more effect on cough. A combination of codeine, atropine, and strychnine is highly efficient as a remedy for cough, for night-sweats, and reflex vomiting. Picrotoxin allays the vomiting which accompanies the cough almost as efficiently as strychnine, and has at the same time decided anhydrotic effect. A resolute patient may suppress cough to a very great extent by an effort of the will. The irritable feeling in the fauces may be allayed by a bit of gum-arabic or candy, or a troche. The officinal troche of licorice and opium, or of morphine and ipecac,* or a troche containing a small quantity of cocaine ($\frac{1}{6}$ to $\frac{1}{4}$ gr.) may be employed in this way advantageously. In the treatment of the *fever* of phthisis, the first and most important remedy is *rest.* Under a mistaken notion of the value of exercise, phthisical subjects, having a high fever, attempt an active out-door life. A very considerable increase of the normal increment of fever takes place when exercise is attempted, and a corresponding diminution when repose is enforced. As a high range of temperature is most injurious, it is necessary to reduce it as much as possible. The most effective antipyretics are antipyrin and quinine, administered in anticipation of the period of pyrexia. Digitalis is too nauseating to be used with advantage, and salicylic acid is more unpleasant in all respects and less efficient than quinine. The most powerful anhydrotic which we possess is atropine. For an adult about $\frac{1}{60}$ of a grain at bed-hour usually suffices ; but, as atropine seems to have a special action on the lungs in caseous pneumonia, it is better to give it in smaller doses ($\frac{1}{200}$ to $\frac{1}{100}$ grain) twice a day. Under its use there is often a remarkable improvement in the condition of the patient, not due solely to the arrest of night-sweats, but to some special property. The combination before referred to is a suitable form for the administration of atropine—with codeine and picrotoxin. Sometimes remarkably good results follow the use of picrotoxin, but it is far from being uniformly successful. If atropine fails, picrotoxin should be tried. Oxide of zinc, with belladonna extract, sometimes does well. Sponging the body with hot water, or vinegar and water, is a domestic remedy, which is refreshing. Remedies for the laryngeal symptoms can be applied directly, the hand being guided by the mirror. Nitrate of silver, carbolic acid, and iodoform are the medicaments most frequently thus used. Atomization and inhalation of vapors are, however, more useful and generally employed. Common salt, potassic chloride, ammonium chloride, corrosive chloride, tannic acid, carbolic acid alone or with tincture of iodine, creosote, ethyl iodide, are the remedies most frequently used in this way. Those used by atomization are dissolved in water, or in glycerin and water, for example, gr. ij of tannin to the ounce of water, the patient receiving the spray in the fauces. Inhalation of vapors, such as ethyl iodide, creosote, tincture of iodine, carbolic acid and tincture of iodine, is of unquestionable utility, and should constitute a part of the treatment in every case. The simplest means suffice to vaporize these agents, and the vapor can be drawn into the lungs by deep, slow inhalations.

The diarrhœa of phthisis is most difficult of control, and for obvious reasons—the tubercular deposit and the subsequent ulcerations.

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* Trochisci glycyrrhizæ et opii, each troche contains $\frac{1}{20}$ grain of opium; trochisci morphinæ et ipecacuanhæ, each troche contains $\frac{1}{40}$ grain of morphine and $\frac{1}{12}$ ipecac.

Opium and acetate of lead, opium and tannin, opium and sulphuric acid, opium and arsenite of potassa, are among the principal remedies. Extract of logwood is highly esteemed by many English practitioners. The author has had better results from Fowler's solution and the tincture of opium than any other remedies (2 gtt. to 10 gtt.) except aromatic sulphuric acid and laudanum (15 gtt. to 10 gtt.). In the treatment of the diarrhœa frequent changes are necessary. A remedy that succeeds for a time will not continue to do so, and hence the resources of the physician are often severely tried.

The requisites of a climate for pulmonary invalids have been briefly stated; they are dryness and elevation. The health-resorts which offer these requisites in the highest perfection are the best. Those of North Carolina, South Carolina, Georgia, the Rocky Mountain regions, California, New Mexico, offer every variety. No change of climate, however, can be beneficial as a rule, after cavities have been formed, unless of slight extent. It is in incipient phthisis that a change to a climate dry, bracing, and elevated, really exerts a curative influence.

HÆMOPTYSIS—BRONCHO-PULMONARY HÆMORRHAGE.

Definition.—The word *hæmoptysis,* which means "spitting of blood," does not indicate the source of the hæmorrhage. *Broncho-pulmonary hæmorrhage* is a correct designation, for this expresses both the nature of the accident and the position of the disease. Bronchial hæmorrhage occurs from some part of the bronchi; pulmonary hæmorrhage consists of two forms—*pulmonary infarction; pulmonary apoplexy*—a hæmorrhage arising from embolic blocking of a branch of the pulmonary artery, the tissues of the lung being displaced merely in the former, but broken up in the latter.

Causes.—Pulmonary hæmorrhage is infrequent at the extremes of life, and is most common from youth up to middle life. It occurs in either sex in about the same ratio. An infarction presents a characteristic appearance of a wedge-shaped portion of the lung infiltrated with blood, and situated at the periphery of the lung, with the base of the wedge outwardly. Infarction is almost always associated with heart disease, in which heart-clots are formed on the right side, and emboli being detached pass into and obstruct a branch of the pulmonary artery. To cause an infarction, the artery obstructed must be a "terminal artery" in the sense intended by Cohnheim*—that is, an artery without anastomoses, and dividing only into the final capillaries. When such a vessel is obstructed, the blood-current is arrested both in front and behind the point of obstruction, in the capillaries and veins, until they are joined by others. Then commences a backward current into the capillaries of the occluded vessel, and into the vessel itself, until they are thoroughly distended with red blood-corpuscles, and hence appear to the eye as a red spot having a wedge shape. In another form of infarction, a diseased vessel giving way, the blood enters a bronchus, and is drawn up into the lobules, distending them. This differs from the other form in appearance ; it is less dark in color, is irregular in outline, and is shaded off into the surrounding normal tint.

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* "Untersuchungen ueber die embolischen Processe," Berlin, 1872, p. 74.

Pulmonary apoplexy is a hæmorrhage which breaks up and infiltrates the lung, and is usually due to traumatism, to gunshot injuries and contusion, to the rupture of aneurisms, to gangrene, etc. Bronchial hæmorrhage arises from primary and secondary causes. The primary causes are of an irritative kind, and induce congestion : prolonged exertion of the voice, mechanical straining, inhalation of irritating gases and fumes, etc. An abnormal weakness of the vessel-wall inherited ; that state of the circulation which exists in the subjects of hæmophilia, the so-called "bleeders" ; the condition of the vessels in young subjects of the strumous type, are factors in the production of bronchial hæmorrhage. The most important of the causes is tuberculosis. As has been stated elsewhere, the initial change in the development of pulmonary tubercle is a proliferation of the connective-tissue corpuscles of the adventitia ; and, although the multiplication is chiefly outwardly, the media and intima are weakened. Hæmorrhage may therefore be an early symptom of tubercular deposit. In the extension of the tubercular deposit a vessel may be invaded at any time. A large hæmorrhage may result from the opening of a vessel by erosion in the process of softening and formation of cavities, or by the development of an aneurism on a vessel in the wall of a cavity. The vessels still pervious are subjected to a much greater pressure by reason of the closure of so many, and hence this increase in the vascular pressure enters into the question of hæmorrhage. The suppression of an habitual discharge has long been supposed to cause pulmonary hæmorrhage, but this is no longer admitted. The menstrual flow may take place vicariously by the bronchial mucous membrane, as it does by various channels. A substitution is very different from a vicarious hæmorrhage.

Pathological Anatomy.—Hæmorrhage may be caused by a diapedesis of red-blood globules, and hence no solution of continuity can be detected under such circumstances. Even when there has been a considerable hæmorrhage, the source of it may elude the most painstaking investigations. If the examination is made immediately after a hæmorrhage, there will be found both fluid and coagulated blood, drawn up into the bronchioles and alveoli, and through the larger tubes. In consequence of violent struggles for breath, in the case of large hæmorrhage, the inspiratory efforts draw up a good deal of blood into the lungs, distending them, so that they overlap the heart and do not collapse. They present a mottled appearance, because of the filling of many alveoli with blood. The mucous membrane of the bronchi may be congested or reddened by patches of extravasation, or of a dull-red by imbibition of blood, or uniformly pale from anæmia, according to the causes producing it and the source of the hæmorrhage. The infarction presents a most characteristic appearance: it is wedge-shaped, with the base outward, and is, when small, just under the pleura; when large, nearer the root of the lung. Infarctions vary in size, from a pigeon's to a hen's egg, or may even occupy a half or nearly the whole of a lobe. They are found more frequently in the inferior part of the lower lobe. If under and next the pleura, they appear as dark-blue masses, projecting somewhat above the general surface of the lung, which just about the infarction is pale and exsanguine, while the pleura is roughened by exudation, confined to the infarction. Sometimes effusion occurs in the pleural cavity, which contains flocculi of membranous exudation, and is red by admixture with blood. When a section is made through an infarction, it appears as a dark, reddish-blue, well-defined mass, from which some dark, reddish-brown liquid and granular matter may be pressed. Fibrinous exudation, distending some of the alveoli, gives to the otherwise smooth surface a granular aspect. At first firm and elastic, the infarction soon becomes friable. The surrounding pulmonary tissue is more or less hyperæmic and œdematous. An infarction may undergo several kinds of change: the blood may disintegrate, the fibrin become granular and fatty, and the corpuscles break up into fat-granules; absorption may take place in part, extrusion in part, and recovery ensue, the elasticity of the lung remaining impaired to some extent. Recovery may ensue in part only: the lobules collapsing and inflammation occurring in the connective tissue, a brownish-red indurated mass remains; or, after an imperfect absorption of the blood and inflammatory exudation, the remaining reddish, pulpy mass solidifies by infiltration with calcareous salts, or, merely inclosed by a limiting membrane, a cyst remains—a process only resembling hæmatoma of the dura mater. Or, again, inflammation may result in suppuration, an abscess forming; or, finally, the whole may become gangrenous. Pulmonary apoplexy not unfrequently forms a blood-mass of considerable size, the blood breaking up the pulmonary elements and diffusing into the surrounding parts, in part coagulating. If next the pleura, this membrane may be perforated, and the blood, entering the cavity, produce a hæmothorax.

Symptoms.—It is but rarely that a hemorrhage occurs in full health without the least intimation of its approach. In this way may the onset of pulmonary disease be announced. Usually there is a sense of heat and oppression of the chest, which those recognize who have experienced former attacks, or there may be general vascular fullness, headache, vertigo, palpitation of the heart, a quick, strong pulse, etc. The signs of pulmonary disease precede the hæmorrhage, in a majority of cases, rather than succeed to it. At the moment the attack is experienced, there are a sudden cough, a warm feeling under the sternum, and a mouthful of fluid, tasting both saltish and sweetish, comes up. Cough now succeeds cough, and with each effort a teaspoonful or more of blood, somewhat frothy, or, if in large quantity, bright—red blood and somewhat darker clots, are discharged. Fven with a small amount of blood, the moral effect of the blood-spitting is so great that much depression, paleness of the face, and a weak pulse result. If the loss be great, there will come on the subjective sensations of fainting, and actual syncope will happen. If the hæmorrhage is great, the blood will come up with a sudden gush, spurting from the nose as well as the mouth. If a fatal hæmorrhage, the blood will pour out of the mouth and nose, there will be gurgling in the fauces, frantic efforts at respiration, a deadly pallor will overspread the face, and, with a general convulsion in which the breathing ceases, all is over, but the heart will beat for a minute longer. The expectoration of blood does not cease with the arrest of the hæmorrhage ; for some days subsequently dark-brownish coagula will be brought up, with some rather viscid mucus. The source of the hæmorrhage may not unfrequently be determined by the moist *râles* heard in the bronchi. The signs and symptoms of infarction have already been mentioned under the head of embolic pneumonia, so that it is necessary only to mention that, when an infarction of sufficient size is formed, the symptoms are sudden dyspnœa and the physical signs of consolidation.

Course, Duration, and Termination.—There are great variations in the amount and duration of pulmonary hæmorrhage. The whole course may be concluded in a few hours. The expectoration may go on during several days, from a tea- to a tablespoonful being spat up each time, and the hæmorrhage in the aggregate amounting to several pounds, causing great depression and a tedious convalescence. In other cases, there may be a number of large hæmorrhages, occurring after an interval of several days, the arrest being due to syncope, and the hæmorrhage recurring when sufficient blood has been made to produce it. Such cases may continue for several weeks, the system being much reduced and the convalescence very protracted. In cases of hæmorrhage with infarction there will follow a period of inflammatory reaction, the expectoration will continue bloody for a week or ten days, and, if the area of tissue involved is small, recovery will ensue, and convalescence will be established in about ten days. The reader is referred to embolic pneumonia for further details in respect to this group of cases. An ordinary croupous pneumonia may be accompanied by considerable hæmorrhage, which occurs with the initial hyperæmia, when the pneumonic process may be confounded with the results of hæmorrhage. The debility caused by pulmonary hæmorrhage is quite disproportioned to the actual loss. A few teaspoonfuls may induce fainting and an unexpected degree of anæmia. Any considerable loss will be followed by pallor, weakness, breathlessness on slight exertion, palpitation, etc., and the restoration of the blood will require several weeks or months. The moral effect of the hæmorrhage and the association of ideas connected with the bleeding are in part responsible for the depression, but more is due to the fact that, in most cases, the system is already enfeebled by a dyscrasia. To this important element is also due the prolonged condition of anæmia—the slow reproduction of the red blood-corpuscles.

Diagnosis.—In every case of doubt, the mouth, fauces, and nares should be carefully examined. Is it vicarious hæmorrhage? The patient is a female, the hæmorrhage occurs at the menstrual epoch, and takes the place of the menses, or nearly so, and no untoward results are experienced, nor does any evidence of pulmonary disease exist. In many of these supposed vicarious hæmorrhages it will be found that the subjects are of the phthisical type, and that, if the physical signs are wanting, there are suspicious rational symptoms. In these cases, it usually happens that the menstrual flow does not return, and that phthisis rapidly develops. Hæmoptysis is to be differentiated from hæmatemesis. In the latter, the blood is black, contains no air, has an acid reaction, is mixed with articles of food, and is vomited; in the former, the blood is bright red, contains air, has an alkaline reaction, and is coughed up, while there is no nausea. If the blood of pulmonary hæmorrhage is swallowed, it will present the characteristics of blood derived directly from the stomach, but the distinction is then made by observing that some of the blood is coughed up, and has the ordinary character of blood derived from the lungs. It should be noted that blood swallowed may pass away with the stools. Hæmoptysis is accompanied by *râles* in the chest, and preceded in the largest number of cases by symptoms referable to the chest; hæmatemesis by symptoms referable to the stomach.

Prognosis.—It is very rare indeed for the life to be put in jeopardy by a pulmonary hæmorrhage. If the patient is much reduced, a severe hæmorrhage may materially hasten a fatal result. Hæmorrhage proceeding from a cavity is more unfavorable than a bronchial hæmorrhage, for the vessel may bleed again and again, since any coagulum, which in other situations might close it, will here be readily detached. The prognosis must be guarded when the subject of the hæmorrhage is much reduced and the quantity lost is considerable. In a case of supposed vicarious hæmorrhage, the probability of a rapid development of the pulmonary lesion should not be forgotten.

Treatment.—The management of cases of hæmoptysis includes the treatment of the hæmorrhage and of the conditions on which the hæmorrhage depends. If the subject be a plethoric one, and there is much oppression from fullness of the vascular system, bloodletting may be practiced, either by venesection or by application of a dozen leeches. These are, it must be admitted, rare cases.

The most effective of the remedies acting dynamically are ergot and barium chloride—the former given subcutaneously and the latter by the stomach. Fluid extract of ergot may be given internally, combined, if desirable, with digitalis and opium—with digitalis if the action of the heart is rapid and excited, and with opium if there is a troublesome cough. Ipecac is, next to those mentioned above, one of the most efficient hæmostatics. Its utility has been disputed on theoretical grounds, but not by those who are practically acquainted with its real advantages. Ipecac produces an exsanguine condition of the lung, and arrests hæmorrhage, also, by the enfeebling effect of nausea on the heart. It is even successful in stopping *post-partum* hæmorrhage. Besides its hæmostatic effect, one advantage of its use consists in mechanically clearing the alveoli of retained clots. Ipecac should not be prescribed in those cases of hæmorrhage from a cavity, the difficulty of keeping a clot in the position necessary to close the vessel being already great. The most suitable form for the use of ipecac is the fluid extract, which may be combined with ergot, digitalis, and opium if desirable. Tincture of veratrum viride may be used with great advantage to keep down the action of the heart. Ice has a similar effect to these dynamical hæmostatics; it slows the heart and contracts the arterioles. It should be applied to the chest, especially to the nape of the neck. The alternate application of heat and cold is usually more effective than the continuous cold. A sponge dipped in hot water can be applied first, then an ice-bag, and so on alternating—the heat remaining in contact but a few minutes, while the cold is kept applied the rest of the time. Absolute rest is an agent of the same kind. The patient should maintain a recumbent posture, and not exert a muscle if he can exercise such restraint. All emotional disturbances should be avoided as well. There are remedies called astringents which are supposed to possess hæmostatic powers, such as tannic and gallic acids, acetate of lead, alum, and the mineral acids, especially sulphuric. These are decidedly inferior to the remedies above named, yet they are freely used, especially the acetate of lead in combination with opium. That they are serviceable, an immense experience confirms, but they do not deserve the very great confidence reposed in them by many practitioners. In cases of debility, characterized by relaxation of tissue, or in examples of the hæmorrhagic diathesis, or in cases of purpura, oil of turpentine is highly useful. Inhalations, by the atomizer, or spray douche, of a solution of Monsel's salt (subsulphate of iron) or the chloride of iron, will sometimes arrest a violent hæmorrhage at once. This undoubted fact is all the more difficult of explanation, since but little, very little, of the iron salt can pass the chink of the glottis, and none of it can reach the point of disease in the lung. Tannin in solution may be employed in the same way, but the iron spray is distinctly better. In administering iron spray great care must be exercised to protect the teeth and the clothing, which may be permanently stained. A mouthful of common salt is a domestic remedy, which may be used until more efficient means are available. Counter-irritants are serviceable. A mustard-plaster or a flying-blister is sufficiently active, or a turpentine liniment, the latter being useful also because of its vapor. Good results may be obtained by inhalation of the vapor of turpentine disengaged for this purpose in those cases appropriate for its internal administration. If the hæmorrhage has shown a disposition to recur, the recumbent position, quietude of mind, and the remedies employed to check it, if not objectionable, should be continued until all possibility of danger has passed.

HYPERÆMIA AND ŒDEMA OF THE LUNGS.

Definition.—*Hyperæmia* signifies an abnormal increase in the blood-supply, which may be active or passive. *Œdema* is usually a consequence of hyperæmia, but it may be due to causes producing general œdema. The term signifies the presence of serous fluid in the alveoli, the intervening connective tissue, the perivascular lymph-spaces, etc.

Causes.—There may be an increase in the amount of blood going to the lungs, the result of increased pressure in the arterial system, from greater force of the heart's contractions, or from narrowing of the arterial field elsewhere, throwing an additional quantity on the lung. Undue exercise of the vocal apparatus in speaking or singing, the inhalation of cold, or very warm air, or the sudden transition from one extreme of temperature to the other, and the inhalation of irritating gases or vapors, are causes determining congestion of the lungs under favoring circumstances. The form and character of the chest and the existence of a constitutional vice or dyscrasia are necessary to bring about the results from the operation of such causes, especially the type of chest and the bodily conformation of phthisical subjects. The ingestion of cold drinks, the body in a warm and perspiring state, will sometimes induce extreme congestion of the lungs. The sudden impact of cold air or cold water on the surface will more surely produce the same result, since a larger surface of the capillaries is made to contract, forcing the blood within. One part of the lung, the seat of a disease obstructing the circulation in it, will necessarily throw on another part an excess in its supply; pneumonia, atelectasis, and obstruction in some branches of the pulmonary artery, are examples. Passive congestion is produced by causes interfering with the return of blood from the lung; mitral stenosis and insufficiency, aortic stenosis and insufficiency, and obstructive lesions maintaining venous stasis, are examples. A weak heart may produce the same result by insufficiency in propulsive power, and hypostatic congestion results from such a state of adynamia that the blood simply obeys the force of gravity. Œdema is a result of congestion, whether active or passive, or a local effect of the causes producing a general dropsy.

Pathological Anatomy.—When the lung is congested it is heavier, contains less air and more blood, and crepitates less than is normal. The color is darker and redder ; on section it is found to contain more fluid in the interstices, more blood flows out from the divided vessels, and the bronchi are injected and filled with a sanguinolent, frothy serum. In chronic cases the congestion is considerable, the color of the affected portions is dark red, almost blackish red ; the interstitial connective tissue is distended with serum, the capillaries are so swollen as to compress the alveoli, almost or quite obliterating the cavity, and numerous extravasations are found through the parenchyma. So firm and dark becomes the tissue of the lung as to resemble the appearance of the spleen, whence the term *splenization* to characterize this condition. In the dependent portions of the lungs of the very adynamic or of aged persons confined to a recumbent position, a serous fluid, having considerable viscidity, exudes, giving to the lung on section a somewhat granular aspect, whence the term *hypostatic pneumonia.* In œdema there is a serous infiltration into the interstitial connective tissue and in the alveoli, which may be sufficient to distend the lung and afford pitting on pressure. On section of the lung under these circumstances, a quantity of serum flows out ; the serum is reddish when there is much congestion associated with the œdema. When œdema of the lung coexists with general dropsy, the fluid that exudes is colorless, and the tissue of the lung is pale. The dependent and inferior portions of the lungs first become œdematous ; thence it spreads to the superior and anterior portions as the fluid increases in amount. As a result of congestion of the passive kind, due to disease of the mitral valve, the lungs generally become denser, more resistant, and are much increased in size. The color, externally, varies from a reddish-yellow to a brown, and on section its texture is found to be firm, to crepitate but little, to exude blood very freely, and not only blood, but, on pressure, to exude a yellowish or brownish fluid. While the general color of the divided surface is yellowish-red or brownish-yellow, there are spots interspersed having a brownish almost blackish color—whence the designation *brown induration.* Some of these brown spots are very dense, and sink in water.

Symptoms.—A sudden and complete congestion of both lungs may be a cause of sudden death. Between this extreme and a simple unilateral congestion of slight extent, there are numerous gradations in the severity of the seizures. In the mildest cases there occur a sense of internal heat, oppression of the chest, some slight difficulty of breathing, a flushed face, a strong, full pulse, beating of the carotids, and injection and brilliancy of the eyes. When the congestion is sufficient to cause universal œdema of the alveoli, the symptoms are formidable. There are great difficulty and extreme rapidity of breathing, a strong sense of oppression, intense anxiety, rapid and violent action of the heart, beating carotids and pulsation in the temples, headache and fullness of the head, a flushed face, a hasty and troubled cough, and expectoration of a frothy liquid which may be tinted with blood.

On percussion the resonance of the lungs is but little altered—slightly diminished, with a tympanitic quality. The vesicular murmur is supplanted by sub-crepitant and mucous *râles*, which are very abundant and very loud. If the alveoli are filled with fluid, the sonority will be still more diminished, and the respiration will have a blowing character approaching bronchophony. If the alveoli are filled to that degree that the oxygen can not reach the blood, accumulation of carbonic acid must take place, and hence there will be blue lips, a livid face, headache, etc. When this condition is reached, there will be still greater anxiety and oppression, the breathing will be shallow and exceedingly hurried, the pulse will decline in volume, and at length will be merely thready and intermittent, the surface of the body will be cold and covered with a clammy sweat, the fingers will be blue and cold, and with the accumulation of carbonic acid there will be increasing somnolence, replacing the extreme restlessness, deepening into coma. With the increasing stupor there will be less and less effort at cough and expulsion of the fluid accumulating in the bronchi, and an increasing difficulty of breathing from this cause. In the cases of passive congestion of the lungs, due to cardiac disease, there are difficulty of breathing, cough and oppression, constantly present, and paroxysms of extreme dyspnœa, in which the patient labors for breath, the face is cyanosed, the extremities cold and blue, the skin cold and covered with a clammy sweat, the pulse, small, weak, and irregular, the jugulars swollen, the mind clouded, etc. The severity of these attacks will be greatly increased if œdema come on suddenly; but if the œdema is gradual in forming, the difficulties of breathing will be slowly augmented, and carbonic-acid poisoning will also be slowly developed. The physical signs in cases of hypostatic congestion will indicate the existence of bilateral lesions if the decubitus is dorsal; or unilateral, if the decubitus is to one side. The sonority is diminished, or dullness with a tympanitic quality exists. On auscultation, the vesicular murmur will be weak, or supplanted by moist *râles*. The difficulty of breathing which arises during chronic Bright's disease is due to œdema of the bronchial mucous membrane—an interstitial œdema and swelling of the terminal bronchi.

Course, Duration, and Termination.—An acute congestion of the lungs may pass through its whole course and prove fatal within a few hours. The usual duration is from three to five days, and the termination may be by resolution, occasionally by hæmorrhage, and rarely by inflammation or pneumonia. The passive form associated with cardiac disease develops slowly, and is subjected to great variations; to periods of improvement under appropriate treatment; then exacerbations. Acute œdema may come on, and prove quickly fatal in acute, or chronic kidney affections.

Diagnosis.—Active congestion is to be distinguished from the stage of engorgement in pneumonia. The points of difference are: in congestion there are no chill, no pain in the side, and not the range of temperature of pneumonia. The subsequent course separates the two diseases more widely. Œdema occurring during hyperæmia is announced by dyspnœa, by the auscultatory signs of the presence of fluid in the terminal bronchi, and by the expectoration of a frothy, serous, and reddish fluid. The hyperæmia of a passive kind produced by valvular lesions is accompanied by rational and physical signs, which make the diagnosis merely a question of the recognition of these signs.

Treatment.—Active congestion in a plethoric subject may demand bloodletting, if not by venesection, by the application of cups or leeches to the chest. A ligature to the thighs applied merely firmly enough to retain the blood in the superficial veins is a useful expedient when the abstraction of blood may seem to be necessary. Counter-irritation in the form of a large mustard-plaster should be applied to the chest, and the feet should be put in a hot foot-bath. As the removal of the fluid in the alveoli and terminal bronchi is of the utmost necessity, an active emetic should be prescribed; of these apomorphine subcutaneously is probably the best, and next, the subsulphate of mercury. Stimulant expectorants should be prescribed to procure the expulsion of the fluid by expectoration. Squill, senega, and serpentaria are appropriate remedies for this purpose. To diminish the viscidity of the fluid, and thus secure its easy expulsion, the iodides, especially the iodide of ammonium, are highly serviceable. The iodide and carbonate of ammonium in sirup of senega is an excellent combination to secure the rapid and easy extrusion of the fluid present. In the œdema of cardiac disease and renal dropsy, digitalis and squill are very important remedies. If the blood is much impoverished, iron is indicated, especially the iodide of iron, which is a rapidly acting and an efficient chalybeate. When there is hypostatic congestion, changes in the position of the patient are very necessary, and the propulsive power of the heart must be increased by stimulants, quinine, and small doses of opium. In the cases of brown induration, the iodide and carbonate of ammonium should be persistently used together, with means to increase the energy of the heart, such as turpentine, eucalyptol, and alcoholic stimulants.

ATELECTASIS.

Definition.—This term means a collapse of the lobules, so that the cavity disappears and the walls approximate. Congenital atelectasis is the state in which the lungs are before being dilated with air (fœtal lung).

Causes.—The congenital condition is simply a failure to distend the alveoli. The whole lung may be in such a state, or only a part of it, in a premature child, or one so weak at full term as to be unable to expand the lungs fully, and hence some of the lobules or alveoli remain in a state of atelectasis. The acquired atelectasis is the collapse of lobules that have been expanded. A terminal bronchus may be closed against the admission of air by a plug of mucus which, acting like a ball-valve, permits the exit, but not the entrance, of air, so that gradually all the residual air is expelled, and then the sides approximate, and the cavity is closed—in other words, it has collapsed. This result is the more apt to occur in the case of feeble, ill-nourished, and ill-developed children, who are attacked with such troubles as measles, whooping-cough, etc. Collapse of lobules—of a large part of a lung, indeed—may be induced by pressure on a bronchus, of an aneurism, of enlarged bronchial glands, tumors, etc. The air remaining in lobules, to which the access of air is cut off, is gradually absorbed by the blood. Direct pressure may also cause atelectasis—such direct pressure as is made by hydrothorax, empyema, hydropericardium, aneurisms, tumors of the thorax, and effusions in the peritoneal cavity, sufficient to push up the diaphragm.

Pathological Anatomy.—Seen from without, those portions of the lung in the atelectatic condition have a bluish-red color, or grayish, and are depressed somewhat below the general surface of the organ. These parts have a greater density than the healthy tissue, and, as they do not contain air, do not crepitate on pressure, and they are tough and not easily broken up. When divided, but little blood flows out, nor do they contain any kind of fluid, and appear smooth instead of granular. When inflated with air, as freshly atelectatic lung can be, an immediate change in color ensues, the lobules become pink, and crepitate on pressure as normal lung. If, however, they continued collapsed, changes of a nutritional kind ensue, and, after a time, dilatation can not be effected. When congenital, this condition is found to exist in the posterior and inferior parts of the lungs, in the apices and anterior borders, and may be limited to individual lobules, or a considerable part of a lobe may be affected. When atelectasis is acquired, usually isolated lobules, or small groups of lobules, are thus affected, they are more or less thickly disseminated through both lungs, and the superficial portions are first attacked, the deeper parts subsequently. This acquired atelectasis differs from the other in that the collapsed parts contain more blood and serum, and hence there is a marked difference in appearance of the affected and surrounding surfaces, since the latter are distended with air, and paler; are, in fact, in the condition of vicarious emphysema. The pleura is usually normal; it may be somewhat congested and thickened. The situation of the collapsed lobules is due to the position of the compressing force. If the force of the compression has not been sufficient to drive all the blood and air out, it is then said to be *carnified;* if all blood and air are excluded, the color is grayish, and the texture is firm.

Symptoms.—In congenital atelectasis, symptoms are produced only in the event that a considerable number of lobules are collapsed, when the chief sign is imperfect respiration. The thorax has but little amplitude of movement, the breathing is rapid but superficial, and the voice is nothing more than a husky whisper. So rapid is the breathing, and urgent the need of air, that a child so affected nurses with difficulty, or not at all. The supply of oxygen being inadequate, carbonic acid accumulates; the lips are blue, the extremities blue and cold, and very feeble, and there are drowsiness, muscular twitchings, and possibly convulsions and paralysis. In the acquired form, the collapse of the lobules is preceded by bronchitis of the finer tubes. When the atelectasis occurs, the difficulty of breathing increases, there is corresponding frequency, and the movements of the two sides may be unequal if there be a limitation to one lung. In inspiration, instead of expansion of the chest in all directions, there is retraction of the intercostal spaces, and of the inferior ribs, due to the fact that the lungs can not be expanded. The significance of the physical signs will depend on the extent to which the atelectasis has proceeded. If isolated lobules only collapse here and there, and the adjacent lobules are dilated (vicarious emphysema), there will be no appreciable change in the sonority. If, however, a group may be collapsed of considerable extent, there will be dullness, but the note will have somewhat the tympanitic quality. The changes on auscultation will depend equally on the amount of tissue in the condition of collapse. The respiratory murmur will be replaced by bronchial sounds if there are a large number of lobules atelectatic. These sounds will also change with the alterations in the affected parts—an increase of the collapse will enlarge the area of dullness; improvement in the local condition and the reëntrance of air will reproduce the vesicular murmur. As very pronounced lesions are associated with the atelectasis, obviously the symptomatology will be very much influenced by them. An important complication arises from the collapse of lobules: the pulmonary circulation is obstructed, the blood accumulates on the right side, the cavities dilate, the venous system is abnormally full, and the arterial system is ischæmic. The results of this state of things are, therefore venous stasis and œdema, the pulse is small, the urine scanty and high-colored, and the skin pale and relaxed.

Course, Duration, and Termination. — The course of atelectasis is that of the malady associated with it. The congenital form, if limited in extent and not associated with a patulous condition of the foramen ovale, may get well. If, however, it is extensive, and especially if the cardiac anomaly exist, life will continue feebly for a short period, and death occur, frequently in convulsions. The acquired condition, when associated with capillary bronchitis and catarrhal pneumonia, pursues two directions : imperfect recovery with damaged lungs, these organs becoming emphysematous ; caseous pneumonia and phthisis. The duration, therefore, becomes indefinite, and the termination that of the associated disease. Acute cases terminating fatally rarely continue longer than one week.

Diagnosis. — Atelectasis is to be distinguished from bronchitis, pneumonia, and effusions in the thorax. As atelectasis is usually associated with bronchitis, the distinction will rest on the evidences of consolidation of the lung, which are not present in bronchitis. There are no real differences between atelectasis and catarrhal pneumonia, since atelectasis occurs more or less in the former ; hence the distinction must rest on the course and behavior, on the locality, and the difficulty of breathing with retraction of the ribs, which occurs in atelectasis and not in catarrhal pneumonia. From croupous pneumonia atelectasis is distinguished by these symptoms, which are peculiar to pneumonia : localized pain, initial chill, high temperature, crepitant *râles*, crisis—and do not occur in atelectasis.

Treatment.—In the congenital disease, the child should be made to cry vigorously, or the lungs should be well expanded by an efficient and careful inflation with condensed air—an ordinary-fire bellows will suffice. The chest should be irritated with mustard and tincture of iodine, the great delicacy of an infant's skin being regarded. Respiratory stimulants are very useful. Belladonna stands first, next strychnia. Suitable nourishment must be given, and stimulants should also be freely but carefully administered. In the treatment of the acquired disease, the accompanying bronchitis is the point to which attention must be directed. The author has witnessed such important results from the use of iodide and carbonate of ammonium, that he must repeat his recommendation of them. They should be given in small doses frequently repeated. By increasing the flow of serum and lessening the viscidity of the tough secretion which occludes the terminal bronchi, the access of air is again secured to the alveoli. Stimulants to the respiratory function are equally necessary as in the congenital form. Belladonna, or, preferably, atropia ($\frac{1}{200}$ grain *ter in die*), turpentine, eucalyptol, copaiba, are very valuable remedies for this purpose. If the symptoms are urgent, emetics must be used to clear the tubes, of which the most effective are apomorphine, subsulphate of mercury, and ipecac. If the strength is reduced, or if the disorder has occurred in a strumous or rachitic subject, quinine, arsenic, iron (syrup. ferri iodidi, ʒ j *ter in die*), and cod-liver oil, are very necessary and useful. Inhalations of compressed air should be practiced as soon as the condition of the patient will warrant it. Inhalations of turpentine-fumes, and of the vapor of iodine or ethyl iodide, are very efficient in removing lingering bronchial lesions.

EMPHYSEMA OF THE LUNGS.

Definition.—As *emphysema* means an infiltration of the connective tissue with air, certain adjectives are necessary to define the position. Pulmonary emphysema is the form of disease meant here. A general emphysema of the connective tissue of the body is produced when a fractured rib, puncturing the lung, permits the air to pass through the injured pleura into the connective tissue. The subject of pulmonary emphysema has been much confused by the variety of terms employed in explanation of the characteristics of the disease. There are two varieties, as regards the part of the lung affected: the *vesicular* and the *interlobular;* the former meaning *alveolar emphysema,* the latter meaning the presence of air in the space between the lobules of the lungs and underneath the pulmonary pleura, whence the terms *interlobular emphysema, sub-pleural emphysema.* When the disease occurs as an idiopathic and independent malady, it is known as *substantive emphysema;* when developed because of another malady, as, for example, the dilatation of the alveoli which occurs because of atelectasis, it is known as *vicarious emphysema.*

Causes.—There is a type of lung, transmitted by heredity, which is peculiarly liable to emphysema. The alveoli are relatively too large and their walls thin; the connective tissue too largely developed; the vascular supply is insufficient; the chest is deep, and the heart lies lower than is normal; and the muscles of respiration are thin and rather weak. Males are more liable than females, because more exposed to the conditions exciting the malady. It is said, but this statement must be regarded as doubtful, that musicians blowing wind-instruments are apt to suffer from it. Various injuries and diseases of the chest which limit the movements of the lungs, as curvature of the spine, pleural adhesions, hydrothorax, tumors, etc., are supposed to produce it. Vicarious emphysema is especially due to attacks of capillary bronchitis and atelectasis in youth and early manhood, or succeeds to whooping-cough and measles for the same reason that bronchitis has led to collapse of lobules, and consequent emphysema of those not collapsed. All of the causes and conditions producing capillary bronchitis are therefore concerned in the production of emphysema. Interlobular and sub pleural emphysema are caused by rupture of acini, usually by such mechanical violence as severe coughing, but there is necessary to this result probably a weakness of the part yielding to such force. Various theories have been proposed to account for the production of emphysema : they may be referred to two groups—inspiratory and expiratory. As, however, original faulty structure may exist, emphysema is produced in such by causes which would not affect healthy lungs. This form or type of structure, which is distinctly hereditary, has been referred to above. In addition to these changes, Freund explains the production of emphysema by a theory which supposes the thorax to be in a condition of fixed dilatation by alterations in the costal cartilages. Although this state of the thorax may sometimes be a cause of emphysema, it can not be so frequently. That the original faulty structure is an important factor in the production of emphysema is certainly true ; but that the respiratory acts of inspiration and expiration have also much influence can not be doubted. A certion proportion of cases of vicarious emphysema are explained by Williams's theory of *negative inspiratory pressure ;* that is, the alveoli appended to unobstructed bronchi dilate in consequence of the increased pressure due to the obstruction and disuse of many tubes. If there exist an hereditary change in the structure of the alveoli, this increased pressure causes them to yield permanently and lose their elasticity. If the inspiratory pressure is thus increased, i. e., by the obstruction to many bronchi throwing a larger volume of air and higher pressure on those admitting air freely, and the expiratory pressure is lessened, there will occur emphysema by atrophy of the alveolar tissue—the theory of Niemeyer. A large proportion of cases are produced undoubtedly by *forced expiration.* In the act of coughing, the glottis being closed, the expiratory pressure is certainly very great, and all the more in the unobstructed lobules, because so many are closed and are in the atelectatic state, throwing the whole force of expiration on a less number of lobules. The result is that the alveoli yield in those parts of the chest not protected by bony walls, at the apex, and toward the root, at the anterior border, in those situations where the emphysematous condition is most decided.

Pathological Anatomy.—Enlargement of the lungs is not always found as expected ; adhesions may prevent the anterior borders coming forward to the median line, or the lungs may be actually smaller than normal by the collapse of many lobules, the occurrence of interstitial pneumonia, and the contraction of the connective tissue. On the other hand, the lungs may fill up the thorax, cover the præcordial space, depress the heart, and lengthen the thorax to the seventh rib by depression of the diaphragm. When the emphysematous lungs are removed from the thorax they do not collapse, and remain full, especially if the bronchi are swollen and filled with viscid mucus, which will prevent the egress of air. The situation of the emphysematous portions will depend on the form. In those cases due to heightened expiratory pressure, the force is expended on the apex and anterior border, and hence here will be found the characteristic changes. In vicarious emphysema, due especially to broncho-pneumonia, the altered portions will exist more widely—at the apex, the anterior border, and along the diaphragm, or they may be very irregularly distributed about the atelectatic points. The appearance of a lung affected with emphysema is peculiar: it is of a pale-red color, the enlarged lobules are little sacs or bladders, not larger than from the size of a pin's-head up to that of a pea, but by the breaking down of the septa between them a number may coalesce, forming a bladder the size of a walnut. When pressure is made, the elasticity of the lung is found to be so much impaired that the pits made disappear slowly or not at all. The tissue of the lung is also very dry and anæmic, and but little fluid of any kind exudes from it on section; but there is much pigment deposited in small, localized collections, and traversing the atrophied tissue in lines, the remains of blood-vessels. On microscopical examination, the walls of the acini are found to be exceedingly thin and attenuated, the septa broken down so that the remains of them merely project into the infundibular area, or disappear entirely.* In some specimens, the intervening connective tissue becomes hypertrophied, so that the walls of the vesicles appear much thickened. In the progress of the atrophic change, the septa between the lobules breaking down, a number of acini are thus converted into a large one. The blood-vessels are from the beginning obstructed, the red corpuscles pass out by diapedesis, and, collected in groups, form the masses of pigment already mentioned, or the blood-globules retained by the arrest of the current and obliteration of the vessels in front form a fine tracery of pigment. The continued pressure sets up a rapid degeneration of the vessel-walls, and they ultimately disappear by absorption, whence it happens that the tissue is dry and bloodless. The obstruction to the pulmonary circulation is ultimately so great that the pulmonary artery and right cavities become greatly distended. Finally, the muscular tissue of the heart undergoes degeneration, granular and fatty. The distention of the veins leads to widespread venous stasis—nutmeg-liver, congested kidneys, and albuminuria, gastro-intestinal hyperæmia and catarrh, passive congestion of the brain, etc.

Symptoms.—The usual history of cases of emphysema is the occurrence of attacks of capillary bronchitis, catarrhal pneumonia, or at least of severe bronchitis at some period in childhood, after which there exists a great susceptibility to colds and frequent attacks of severe catarrh with difficulty of breathing. After puberty the difficulty of breathing is found to be more decided; bronchial catarrh is not then a matter of cold weather and attacks of acute cold, but is constantly present. In other cases, after whooping-cough, or measles, a troublesome cough, bronchial catarrh, and shortness of breath come on, and steadily increase. If such attacks have occurred in youth, by the time of puberty the emphysema is pronounced, and the chest has assumed the peculiar "barrel-shape," characteristic of this disease. In still another group of cases, the onset is gradual, and the emphysema is the outgrowth of years of bronchial catarrh, the fully developed emphysema not being attained until the middle or after period of life. In which mode soever emphysema manifests itself, the difficulty of breathing is the most pronounced symptom. In all attempts at active exercise, mounting stairways, ascending heights, etc., the breathing is embarrassed. Even before the patient is conscious of his pulmonary defects in this direction, a good observer will note the frequency and imperfect expansion of the thorax. The shortness of breathing is dependent on several factors: the diminution in the number of capillaries has an effect in this way by the lessening, which the loss of vessels involves, of the oxygenation of the blood, so that increasing frequency of respiration is compensatory of this deficiency. Again, depression of the diaphragm renders additional efforts on the part of the inspiratory muscles necessary, and hence this adds to the difficulty of carrying on respiration. More important than these is the loss of the elasticity of the lung, which requires that the muscles of expiration shall take up the labor of expelling the air, which they accomplish slowly and with great effort. This expiratory insufficiency involves another difficulty—the residual air in the acini is not displaced, and hence can not furnish oxygen to the blood. The concurrence of these several factors produces the most obvious objective symptom in emphysema—the embarrassed respiration. Both inspiration and expiration are embarrassed; all the muscles, auxiliary as well as ordinary, are engaged in inspiration and expiration, but the movements of the chest are very slight notwithstanding the labor, and a constant and distressing sense of the need of air is experienced; the cervical muscles are rigid and prominent, the head erect and forward to permit the easy entrance of air and to facilitate the action of the muscles; the shoulders elevated; the veins of the neck enlarged and dilated, and the face more or less cyanosed. A peculiar configuration of the chest is brought about by emphysema, which has existed for some time in young subjects. The chest becomes round; the intercostal spaces wider; the vertical diameter elongated. As the emphysema may be limited to one part, the changes in the shape of the chest will correspond. The departure from the normal consists in a circumscribed prominence more frequently on the left than the right side; above the clavicle, or between the clavicle and nipple, or, during coughing, the lung pushes the parietes of the chest forward at these points, producing a soft, elastic, and resonant swelling. The physical signs are very instructive. On inspection, the character of the respiration, the movements of the accessory muscles, and the extremely small excursions of the thorax in breathing are readily ascertained. On palpation, the vocal fremitus is diminished, the apical impulse is feeble, and the epigastric pulsations are increased. The heart is found to lie lower down than in the normal thorax, and the liver is also pushed lower, both due to the enlargement of the lungs in the vertical diameter. On percussion, the sonority is increased over all the emphysematous portions, and, when the whole lung is involved, extends down to the seventh or eighth rib in front, and behind to the twelfth rib in extreme cases. The hepatic dullness may not begin until the inferior margin of the ribs is reached, and even when hypertrophy exists the area of cardiac dullness is much narrowed and may not exist at all when the emphysema is extreme. On auscultation over all parts returning a resonant percussion-note, the vesicular murmur is weakened, and may entirely disappear over the lungs; and the bronchial sounds, which are audible at the root of the lungs posteriorly in the normal state, may also disappear. In other cases, the vesicular murmur, whether enfeebled or not, is changed in character; on inspiration it becomes rough, rude, sibilant or crackling, due to the entrance of air into the dilated and inelastic lobules, and expiration is prolonged and rough from the same cause. Expiration is usually inaudible, but an expiratory sound may be due to an accompanying bronchitis, to narrowing of the bronchioles by swelling of the mucous membrane, whence the sound has a rather sibilant character. The accompanying bronchitis, which is usually quite extensive, produces various moist sounds—sub-crepitant, mucous, and sub-mucous *râles*, which are not necessary to emphysema. The sounds of the heart audible in the mitral and aortic area are in emphysema less distinct than in the normal state, while in the pulmonary and tricuspid area they are well defined, the pulmonary second sound being sharply accentuated.

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* Thierfelder, "Pathologische Histologie," 1. Lieferung, Tafel vi.

Course, Duration, and Termination.—Emphysema is an essentially chronic malady. Beginning often years before any great difficulty of breathing is manifest, it pursues a course which in its mildest form may continue during an ordinary lifetime. The least extensive cases may continue with little interference in the duties of life for many years, but the position is far different with those examples of emphysema occupying a large part of both lungs. In a pronounced case, beginning in one of the modes already described, there are constant difficulty of breathing, and cough and expectoration due to an attendant bronchitis. On taking a bronchial cold, to which they are extremely liable, or on making some sudden muscular effort, the difficulty of breathing is greatly increased, they labor to get breath, are blue in the face, sweating with their exertions, and unable to lie down. After some hours, or a day or two, the paroxysm subsides, and they are back again in the former condition, except each attack increases a little the existing mischief, the breathing is a little more embarrassed, and there are more cough and expectoration. The paroxysms of asthmatic difficulty of breathing increase in number and frequency, until after some years there is no period of partial relief. Meanwhile, the obstacles to the pulmonary circulation increase: dilatation of the right cavities of the heart and stasis in the venous system occur; the liver swells with venous hyperæmia; the gastro-intestinal mucous membrane also is hyperæmic, and is affected with catarrh; the liver is congested, and the urine becomes albuminous. General dropsy now comes on, fluid accumulates in the peritoneal cavity also, but to a less extent in the pleura. The presence of fluid in the two cavities adds to the difficulty of respiration, and now the patient can get breath only as he sits up, leaning somewhat forward. This position increases the accumulation of fluid in the legs, which become blue, cold, and very painful; the skin yields, blisters form, and, giving way, an ulcer is established from which serum continuously exudes. Such is the course of a well-defined case. Although all are not so severe, yet when emphysema occurs in an adult it is a permanent condition. It is probable that a slight amount of emphysema in a child may get well, but usually the first changes in childhood are the initial of a long series, and continue. Death may be due to the rupture of some of the dilated cells and the formation of an extensive interlobular and subpleural emphysema. The termination is often by some intercurrent disease, as catarrhal or croupous pneumonia, cerebral hæmorrhage, or paralysis of the heart. Notwithstanding the unpromising nature of the disease, all do not proceed regularly from bad to worse. Periods of improvement may take place, and the difficulty of breathing almost disappears, to return again, however, on the occurrence of a bronchial attack or some other disturbance. The cases are, as a rule, more severe in winter than in summer.

Diagnosis.—The diseases with which emphysema may be confounded are bronchitis, bronchial asthma, catarrhal pneumonia, pneumothorax, aneurism of the arch of the aorta, and cardiac diseases, with spasmodic difficulty of breathing. From bronchitis, it is distinguished by the presence of those signs characteristic of emphysema, as difficulty of breathing, increased sonority of the chest, changes in the shape and size of the thorax, and by the disturbances of the circulation and dropsy; from bronchial or spasmodic asthma, by the fact that in the latter there are no alterations of the chest, and the difficulty of breathing is occasional and spasmodic entirely; from catarrhal pneumonia, by the history, by the localization of the affection, by the changes in the chest, and by the subsequent course ; from pneumothorax, by these considerations : pneumothorax is sudden, almost always unilateral, the chest much distended, the intercostal spaces prominent, the heart is displaced to the other side, succussion is present if there is fluid, which is usually the case. In aneurism there is dullness instead of increased sonority over the site of the aneurism, and no change elsewhere, and the difficulty of breathing is due to paralysis of the vocal cord, which may be seen, and to pressure on nerve-trunks. In heart-disease the area of dullness is not only present but usually increased, and the apex-beat is normal or increased, while the form of the chest and the sonority are not affected.

Treatment.—As we have to deal with an incurable disease, our treatment must be largely palliative. For the asthmatic attacks there is no remedy so efficient as the subcutaneous injection of morphine and atropine ($\frac{1}{6}$ morphine and $\frac{1}{120}$ atropine). Care must be exercised lest the morphine-habit be formed, as it is apt to be under these circumstances, and hence the injections should always be practiced by the physician, and reserved for occasions of great distress. A single injection may arrest a paroxysm, but the dose may be repeated as necessary, rarely more frequently than once in six hours. Next to the injection of morphine, most relief is afforded by full doses of iodide of potassium alone, or combined with the bromide. From fifteen to twenty grains of the iodide, and forty grains of the bromide, every two, three, or four hours, according to the urgency, may be prescribed. Chloral, which affords great relief, is very unsafe in old cases with dilated right cavities ; if given under any circumstances, it should be combined with morphine and atropine to prevent the depressing effect on the heart. A combination of morphine, chloral, and atropine is an exceedingly serviceable combination for the relief of the difficult breathing. Besides these agents, narcotic fumigation may be practiced. Pastils of belladonna, stramonium, tobacco, opium, eucalyptus, etc., may be burned, and the fumes inhaled. Such pastils are always much used by these sufferers, since they procure in this way ready and considerable relief. Inhalation of ethyl iodide is a measure of great utility, and is free from danger. As the accompanying bronchitis is an important element in these cases, measures are necessary to relieve it. The best results are obtained from copaiba, turpentine, and eucalyptol, given in conjunction with the iodide of ammonium. Excellent results are obtained from the combined administration of iodide of ammonium and arsenic, continued for some time. It is well known that arsenic increases the depth and volume of the respiration and promotes the nutrition of the lung, and the iodide is an effective remedy for the bronchitis. In these facts we have an explanation of the utility of the combination. When the bronchial secretions are insufficient, small doses of tartrate of antimony are very useful, and give great relief. Quebracho, which has lately been brought forward as a remedy for dyspnœa, is a valuable palliative. Atropine is a remedy of great power, and has an influence over the lung, increasing the respiration and promoting the nutrition of the organ. It may distress if there is a lack of bronchial secretion, but usually the opposite state obtains, and consequently atropine can be given, as it ought to be, under these circumstances, in small doses twice a day for a long period. Of all the means hitherto proposed for the relief of emphysema, nothing has approached compressed air in effectiveness. Indeed, this is the only scientific remedy which has as yet been brought forward for the treatment of emphysema. The chamber into which air is pumped until a pressure of one and a half to two atmospheres is obtained is the best arrangement, but unfortunately they are available but in a few places. The portable apparatus of Waldenburg is convenient, easily managed, and produces good results. The object of compressed air is to relieve the breathing by supplying more oxygen, and it effects an equalization of the blood in the two systems by redistributing the pressure. By retarding the breathing and the action of the heart, the contractions are firmer, and the cavities are better emptied. The improved condition of the blood, the result of a better supply of oxygen and increased excretion of carbonic acid, induces a better state of digestion and assimilation. By breathing compressed air, the pressure is transferred from the venous to the arterial system, and while the amount of blood on the right side is diminished, on the left it is increased. The good effects of breathing compressed air are enhanced by expiration into rarefied air, which of course has the effect to draw the blood into the lungs. "Expiration into rarefied air is the specific mechanical antidote to emphysema." * The inhalation of compressed air or of oxygen may be used as a palliative to relieve the attacks of spasmodic difficulty of breathing.

The treatment of the dropsy requires a nice adjustment of means to the object. Much can be accomplished by acting on the skin and kidneys. If the heart will bear it, pilocarpine may be employed to act on the skin. Hydragogue cathartics can be given at the same time, of which the pulv. jalapæ comp. is best. A teaspoonful or two should be taken in the early morning, and pilocarpine in the afternoon. If the desired results can not be thus attained, free diuresis may be attempted while the hydragogue is also administered. Basham's mixture is an excellent combination, containing as it does a chalybeate with a saline. Niemeyer's prescription of vinegar of squill, with bicarbonate of potassa—thus forming acetate of potassa—is a good diuretic. There is no more certain diuretic than bitartrate of potassa, and it may be combined with infusion of juniper and squill. A weak solution of cream of tartar may be drunk *ad libitum*. Infusion of digitalis may also be given; but as so much obstruction exists in the lung, and as there is also ischæmia of the arterial system, its use is doubtful.

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* "Die pneumatische Behandlung," etc., Dr. L. Waldenburg, Hirschwald, Berlin, 1875, p. 302.

GANGRENE OF THE LUNG.

Definition.—*Gangrene* is the same morbid process, whether occurring in the lung or elsewhere. Gangrene of the lung, therefore, means the death and decomposition of a greater or less portion of the lung-tissue.

Causes.—Sex exercises an important influence, since somewhat more than two thirds of the cases occur in men. Although it may happen at any age, it is more common from puberty to middle life. A lowered condition of the vital forces, such as is produced by abject poverty and its attendant miseries, seems necessary to the result. Interruptions to the blood-supply, as elsewhere, may induce gangrene. Thus it occurs in cases of pneumonia, hæmorrhagic infarctions, catarrhal pneumonia, etc.; but a depressed bodily state is necessary, such as exists in drunkards who are ill fed and exposed to cold and wet. Gangrene may be due to the so-called blood-diseases—as typhus, diabetes, small-pox, measles, etc.—but a low state of the tissues or a depressing cachexia must coincide, the lung becoming the seat of the morbid process because invited by a local malady, such as pneumonia. The deposit in the lung of septic and decomposing materials, as septic or infective emboli, will set up a destructive inflammation terminating in gangrene. Putrefactive decomposition in the neighborhood of the lungs, the penetration of the organ by cancer-masses, or the lodgment of foreign bodies, may give rise to a gangrenous inflammation. Lastly, gangrene may be due to traumatism, or to penetrating wounds of the chest.

Pathological Anatomy.—Gangrene may attack any part of the lung, but the upper lobe is more often the seat of it than the inferior. It occurs in two forms, of circumscribed, of diffused—the former being well defined and strictly limited, the other not separated by any defined border, but spreading into the surrounding lung-tissue. The circumscribed form attacks by preference the outer portion of the lung, and may or may not include the pleura. There may be several of the gangrenous spots, which vary in size from a pea to an orange, or even larger, and they occur rather more frequently in the right lung. The borders are clearly marked, the surrounding tissue being hepatized or œdematous. According to the time at which the masses are examined, they are firm, dry, almost black or soft, diffluent, greenish, or brownish, decomposing and offensive masses traversed by large vessels not destroyed, and by bronchi, opened by ulceration, through which the liquid and softened *débris* are discharging. Gradually sloughing off after evacuation by the bronchi, there may be an attempt at repair, the spread of the decomposition being prevented by the formation of a dense, tough, and rather hyperæmic connective-tissue membrane. A complete recovery can only occur when the gangrenous mass is small and communicates with a small bronchus. The membrane lining the cavity, formed as just described, pours out a quantity of ichorous pus, which serves to spread the morbid process. When the cavity is small enough to close and heal, granulations are thrown out, the walls approximate, and healing takes place, a cicatrix remaining. The ichorous pus poured out from the so-called pyogenic membrane sets up a destructive inflammation of the bronchial mucous membrane, which softens and is detached, and excites attacks in the dependent parts of the lungs of broncho-pneumonia, which pursue the same course. If situated at the periphery of the lung the softening may involve the pleura, and the decomposing materials be discharged into the pleural cavity, exciting a violent pleuritis and a pyopneumothorax, if a bronchus is at the same time opened. It is a remarkable fact that a limiting pleuritis may confine the inflammation to a small extent of the membrane, perforation of the thorax ultimately ensue, with a termination in recovery. In a few cases the pus has dissected downward along the sheath of the psoas muscle and opened externally at the groin. The diffused form may, as has been shown, arise from the circumscribed by an extension of the morbid process through the distribution of the ichorous pus from a gangrene cavity. But the diffused form usually has its origin in an inflammation proceeding from a gangrenous cavity, or from a case of purulent infiltration of pneumonia. The tissue affected with the gangrenous inflammation rapidly breaks up into shreds of decomposing materials, infiltrated with a brownish or blackish fetid fluid, and the morbid process spreads into the surrounding tissue, hepatized and œdematous, without any defined boundary. In a short time much of the upper lobe may be in a gangrenous state, and the whole of it, indeed, may be involved. In both forms the spread of the gangrene may be too rapid to permit the vessels to be closed, and hence there may be formidable or fatal hæmorrhage. Metastatic abscesses may form in various organs, from infective emboli proceeding from the veins of the gangrenous parts.

Symptoms.—Gangrene of the lung being usually a secondary disease, the symptoms proper to the gangrene are obscured by the associated malady; and there are great variations at different periods. Before communication is established with a bronchus, when the diagnosis is rendered certain by the character of the expectorated matters, the only symptoms are, a sudden depression of the powers of life, changes in the character of the existing fever, and a very high range of temperature. The symptoms become characteristic only when the sputa contain the materials of the gangrenous decomposition. The sputum is a sanguinolent, sanious, or sero-mucus fluid, of brownish dark-green, or even blackish tint, having a horribly fetid odor, compounded of decomposing animal matter and fæces, and so sickening that the patient himself as well as those about him is nauseated by it. That the odor is due to foul gases is evident from the fact that the breath on forced expiration is full of the odor, and the sputa allowed to stand cease after a time to have the smell. The odor may precede the expectoration, and may disappear for a time, to reappear again. The sputa on standing separate into three distinct layers: the uppermost, frothy, of a dark, greenish-yellow color, is composed of muco-pus chiefly; the middle layer is sero-albuminous and translucent; the lowest layer contains a sediment, greenish or brownish in color, with yellow or brownish flakes and masses of decomposing lung-tissue. Again, the sputa may be made up largely of black blood, in a decomposing state (Hertz). Chemically, the sputa have an alkaline reaction, and contain valerianic acid, the fat acids, leucin and tyrosin, triple phosphate, and other products of decomposition. During the process of development of the gangrene, the symptoms indicate the existence of a grave disorder. The elevation of temperature may be very considerable, but the thermal line is that of septicæmia: irregular chills, high fever, and profuse sweats. The complexion is fawn-color, livid, the expression anxious, the face sunken, the skin relaxed, the pulse quick and feeble, and the respirations are hurried and catching. There is usually severe pain in the side, and the decubitus is toward and on the affected side. There is an incessant and very painful suppressed cough. Copious pulmonary hæmorrhage may and usually does take place, started by the coughing. The fetid expectoration is apt to be swallowed, and excites by its presence nausea, vomiting, and diarrhœa, but the absorption of putrid matters and the congestion of the portal circulation will also cause watery and fetid stools. The operation of these causes rapidly exhausts the vital powers, and the patient lapses into a condition of profound adynamia. The physical signs are such as pertain to changes in the density of the pulmonary tissue. On percussion, the sonority of the chest is lessened in proportion to the extent of the solidification, but, as there is more or less pulmonary tissue still pervious to air about the gangrenous portions, the dullness has somewhat the tympanitic quality. On auscultation, coarse *râles*, mucous and submucous, are audible, and there are bronchial breath and bronchial voice. After the softening and extrusion of the gangrenous portions, the physical signs will correspond, and the symptoms of a cavity will be present.

Course, Duration, and Termination.—The course of the disease is so largely affected by the morbid condition on which it is ingrafted that no defined plan can be laid down. The circumscribed form is slower in development, and the symptoms are less formidable, than the diffused, and its duration is therefore longer. In those cases which tend to cure by the extrusion of the gangrenous mass through a bronchus, or by establishing a fistulous communication externally, the duration is protracted, and not to be expressed with definiteness, because so much depends on the vital resources, and on the size of the gangrenous patch. The cases of partial recovery in which there is a cavity lined by a pyogenic membrane continue for months; but every now and then fresh inflammation arises, more tissue is destroyed, until death finally ensues. The usual termination is in death, after two or three or even six weeks of the circumscribed form, and in a week or two of the diffused form. Certain accidents may occur which will materially abbreviate either, as hæmorrhage, perforation of the pleura, etc. The causes of death are various—pleuritis, peritonitis, hæmorrhage, exhaustion, etc. Perforation of the pleura may cause death by the intermediation of pyopneumothorax, sudden distention of the cavity, severe dyspnœa, and collapse; or it may cause a fistulous communication, emphysema of the connective tissue, and exhaustion, the fistula discharging ichorous serum and the foul-smelling products of gangrenous decomposition. Perforation of the diaphragm and purulent peritonitis may be a cause of death. The prognosis is, of course, exceedingly grave.

Diagnosis.—It must be obvious that a diagnosis of gangrene of the lung is not possible when the mass affected does not communicate with a bronchus. Fetor of the breath is, of course, the first indication, but this is not pathognomonic by any means. As the pus in cavities and of dilated bronchi may by decomposition become fetid, and as bits of decomposing lung-tissue are cast off in the sputa, fetor of the sputa as a means of diagnosis must be accepted with limitations. The diagnosis, under these circumstances, must rest largely with the clinical history, the severity of the symptoms, and the duration. Those familiar with the character of the odor in gangrene will recognize its penetrating power and intensity, as compared with the much feebler odor in putrid bronchitis and in bronchiectasis. All of the symptoms in gangrene of the lung are much more active and severe than are those of bronchitis. In gangrene, further, there are present the physical signs of pulmonary disease, which are absent in bronchitis. The differentiation of fetid sputa from a cavity in phthisis, from gangrene, is more difficult, but the greater intensity of the odor in the latter and the appearance and composition of the sputa will serve to distinguish between them; but, as cavities are present, the history and behavior of the two maladies must be taken into consideration.

Treatment.—To maintain the powers of life by the free administration of spirits, small doses of opium and quinia, and such aliment as beef-juice, egg-nog, etc., is the leading indication, to which all specific treatment must be subordinated. Excellent results have been obtained from turpentine (gtt. v) every two hours; but still more from eucalyptol, which has been very much extolled recently. Eucalyptol is most easily taken in perls (♏ v), but it can be made tolerable in an emulsion. Benzoic acid, thymol, and carbolic acid, especially the last named, are very useful in correcting fetor, and also play the part of antiseptics, being eliminated largely by the lungs. Acetate of lead is the remedy most approved by Traube. Inhalations should be practiced with those remedies, such as iodine, which may diffuse by volatilization, and with oxygen, which relieves the dyspnœa and improves the blood. Iodine, or the tincture, may be vaporized by a gentle warmth, and the fumes gradually introduced into the air the patient is breathing. Ethyl iodide, oil of eucalyptus, and turpentine, are among the most useful remedies to be administered by inhalation.

CARCINOMA OF THE LUNG.

Pathogeny.—Cancer of the lung is usually secondary, and very often succeeds to cancer of the breast removed by amputation. It may be primary, but rarely so. While cancer of the lung as a secondary disease is more common in women, primary cancer of the lung is more common in men. It is a disease of advanced life, and is extremely rare before forty ; nevertheless, a case has occurred at twenty-five. The form of cancer which attacks the lungs is usually the soft and rapidly growing variety known as encephaloid, and it occurs in two forms—in a distinct body or mass, and diffused through the tissue of the lung. In either case it presents the appearance of a yellowish-white, homogeneous, rather firm material, looking like brain-tissue which had been somewhat hardened—hence the name. When a mass is divided, a quantity of whitish, albuminous-looking fluid may be pressed out, and this fluid is called *cancer-juice*. Sometimes this cancer-juice may be found in cyst-like nodules, or in delicate canals, whitish in appearance, accompanying the lymph-canals. Cancer may occur in any part of the lung ; when primary, in about two thirds of the cases in one lung, and when secondary in both, usually. The right lung is more frequently the seat of cancer, in so large a proportion as two to one. The distribution of cancer varies. In the primary form it occurs in nodules, from a pea to an orange in size, or there may be a great number of the smallest nodules, or a diffused infiltration involving a part or the whole of a lobe, even of two lobes. When it forms a distinct tumor of considerable size, the neighboring parts may be compressed : the lung may atrophy from pressure ; the bronchi may be encroached on and closed, or the cancer elements may enter and fill them ; blood-vessels may be impinged on, their lumen obliterated, or they may ulcerate and hæmorrhage result. The bronchi, trachea, and great vessels may be so far obstructed as to interfere with their functions respectively. The bronchial, tracheal, cervical, and axillary glands may be enlarged from simple adenitis, or from cancerous infiltration. The pleura is usually invaded ; there may be an effusion into the cavity, or adhesions unite the two surfaces, and the cancer elements may make their way to the surface as nodules, or in thin plates. A large cancerous mass may displace organs, push the heart aside, and force the liver and spleen downward.

Symptoms.—When the cancer forms a tumor, the symptoms produced by it are dullness over the place occupied, increase of the vocal fremitus, and bronchial voice and breath sounds over the dull area. These sounds may have the cavernous character if the cancer-mass surrounds, without compressing, a large bronchus. Also, a large artery, impinged on by the tumor, will give forth a distinct systolic *bruit*, which may be mistaken for aneurismal *bruit*, unless it is recognized that there is but one center of pulsation (the heart) in the chest. If the growth be so situated as to press on a large vein, there will be present œdema of the head and face, or of one side; if it press on the recurrent laryngeal, spasm of the glottis, a peculiar cough (croupy), and difficult breathing, or, if the pressure be long continued, paralysis with its usual consequences, will result; if other nerve-trunks are impinged on, there will be deep-seated pains in the thorax, often of an excruciating kind, and there may be paroxysms simulating angina pectoris. The symptoms become more complex and difficult of interpretation, in cases of diffused or disseminated cancer. There are present the signs of consolidated lung-tissue on one or both sides. There are no adventitious sounds, but the respiration has a rather blowing character in some situations; in others, that of bronchial voice and bronchial breath. The diagnosis rests on these facts: all acute diseases are excluded, as this is comparatively slow in development and is free from fever; it can not be chronic pneumonia, as there is no localization of the deposits; from tuberculosis it is separated by the absence of fine crackling, and by the fever-movement; and, lastly, some indurated glands may be found in the neck or axilla, and possibly the traces of a former operation. There will be some difficulty of breathing if the deposits are extensive, and a dry, hard cough; but there may occur, finally, rusty-colored, semi-transparent, gelatinous expectoration. The difficulty of breathing depends on different conditions from those which obtain in the other form. In this case, the degree in which the air-space is encroached upon determines the amount of dyspnœa; in the other, compression of bronchi, or trachea, or displacement of the lung, affects the breathing. The character of the cough is very different, according as it is due to deposits in the lungs, to pressure on a bronchus, to irritation of the recurrent laryngeal, or pneumogastric nerves, etc. Besides the symptoms produced by and due to the presence of the cancer in the lungs, there is soon developed the cancerous cachexia, which is manifested by the following symptoms: progressive emaciation, weakness and sense of fatigue, a weak, small pulse, a peculiar earthy or fawn-color tint of the skin, pearly sclerotic, anorexia, œdema of the ankles, etc. The rate of decline due to the cancer deposits is accelerated by the harassing cough, the dyspnœa, the dysphagia, and the pain. As the cancer extends, all of the rational symptoms increase in severity, and the physical signs more clearly indicate the diffusion of the cancer elements through the lungs, or the enlargement of the tumor.

Treatment.—This must be directed by the symptomatic indications. Anodynes to relieve pain and support for the increasing weakness are the measures necessary.

HYDATIDS OF THE LUNGS—ECHINOCOCCI.

Definition.—Hydatids found in the lungs are the intermediate or larval condition of the *tænia echinococcus*—the tape-worm of the dog—and are therefore designated *echinococci.* The cysticercus cellulosus, the larval state of the *tænia solium*, is very rarely, if ever, found in the lungs. Echinococci migrate from the intestines and take up their abode in the lungs. Each cyst contains the embryo—the scolex with its four suckers, and row of hooklets, inverted and contained within its cyst.

Dermoid cysts are rarely found in the thorax, but they should not be confounded with echinococci.

Pathological Anatomy.—Hydatid cysts usually exist in the parenchyma of the lungs, but sometimes develop in the cavity of the pleura, or they may be present in both at the same time. They are found in the inferior lobe, and chiefly on the right side. Often, the intra-thoracic cyst is a solitary hydatid, which fills the cavity, distending and enlarging the chest on that side, pushing out and widening the intercostal spaces, compressing the lung against the root and the spinal column, and forcing the heart downward or to one side; and depressing the liver or spleen. If the cyst is large, the pleural surfaces may be united and the cavity obliterated. Adhesions are often formed to a bronchus, which may be perforated and a cure effected by discharging the parasite by expectoration. The cavity which remains contracts and cicatrizes. In other cases the parasite is not discharged, but sets up an inflammatory induration about it, which excites fever, cough, and expectoration, that ultimately exhaust the patient unless carried off by some intercurrent affection. Rarely do hydatids come into relation with the vessels of the thorax, but a vessel may be invaded, with results determined by its size. Habershon * reports a case of a youth of seventeen in whom repeated hæmorrhages occurred, from an opening into a branch of the pulmonary vein, produced by "ulceration at the seat of the hydatid cyst." In this case tubercular disease followed the troubles due to the hydatids. Sometimes the cysts attain sufficient volume to cause death by suffocation. In other cases death is produced by atrophy of the inferior lobes of the lungs. In a larger number of cases, pneumonia and gangrene of the lung, induced by the presence and pressure of the hydatids, are the cause of death. The length of time hydatids continue in the lungs is measured by years. The ordinary duration is two to four years.

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* "Guy's Hospital Reports," third series, vol. xviii, 1872-'73, p. 373.

Symptoms.—The cysts must attain a sufficient size to interfere with function before symptoms are produced. More frequently than in other situations, hydatids of the lungs give rise to pains which may be felt in the back, in the side, or in the epigastrium. The pain is severe, persistent, and is somewhat paroxysmal, and its situation may indicate the seat of the mischief. The decubitus is on the back or on the affected side. The most marked as well as the most constant symptom is dyspnœa, which is always present in a moderate degree unless the cyst is very voluminous, and there occur also violent paroxysms, in which the breathing is suffocative. The cough is dry, or accompanied with a little expectoration, unless the cyst communicate with a bronchus, when the cough is incessant and the expectoration enormous, consisting of a serous liquid or earthy and calcareous masses, filled with the *débris* of hydatids. Sometimes the expectoration is fetid, from gangrene, or bloody. Small hydatids of the volume of a pigeon's-egg may be expected, but usually fragments and hooklets. The expectoration takes place at intervals sometimes of weeks or months; then a great mass may come up, almost suffocating the patient.

The physical signs will depend largely on the volume attained by the cysts, their number and situation. There may be seen, on inspection, an enlargement of the affected side, dilatation of the intercostal spaces, and displacement of the heart or of the liver, or of both. Fluctuation or the purring tremor will be felt only if the cysts are protruding through the chest-walls, and if a number of daughter-vesicles are contained within the parent-cyst. On percussion, there will be dullness according to the space occupied, and increase of resistance, commencing below the clavicle, over the inferior lobe. The vocal fremitus is diminished. The vesicular murmur is absent, replaced by bronchial voice and bronchial breath. Egophony may be audible. The signs of a cavity will be present when the cysts are expectorated.

Course, Duration, and Termination.—The origin and early development of echinococci of the lung necessarily escape detection. It is only when they are large enough to interfere with neighboring parts that symptoms are produced. The whole course is usually completed within four years, sometimes earlier, if the opportunity for free discharge exists by an opening into a bronchus. In forty cases of which Davaine* has given an account, there were fifteen recoveries and twenty-five deaths, the termination by expectoration of the hydatids occurring in twelve cases. Of the twenty-five fatal cases, twelve or thirteen occupied the inferior lobe, and five or six the upper lobe. In another collection of cases quoted by Davaine, of sixty-two terminating in recovery forty-five recovered by the expectoration of the cysts, and seven by puncture of the chest, expectoration also occurring. The proportion of cures to cases in the last-mentioned collection was sixty-two to eighty-two. The termination by death is therefore more common than recovery. Death is due to a variety of causes—to exhaustion from profuse purulent expectoration, hectic and marasmus, to tuberculosis, to hæmorrhage, to gangrene, to pleuritis, etc.

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* "Traité des Entozoaires," *op. cit.*, whose account I have closely followed in this subject.

Diagnosis.—There are no well-marked distinctions between hydatid cysts and pleuritic effusion, as regards the physical signs, but they differ widely in history. Pleuritis begins by a violent pain in the side, chill and fever, the effusion following in a short time. Echinococci very slowly develop, and the symptoms of effusion are not produced until after many months. Puncture and examination of the fluid for the characteristic hooklets may be required, to determine the question at issue. When expectoration of echinococci or of fragments takes place, there can be no doubt left.

Treatment.—When the existence of hydatid cysts is ascertained, there should, if possible, be made a free opening to permit their evacuation. Puncture and withdrawal of fluid will arrest their growth, but, as decomposition, suppuration, even gangrene may result, the extrusion of the cysts should be procured, if possible. Free opening into the cyst cavity, and drainage with antiseptic precautions, has proved very effective in the hands of Dr. Fenger, of Chicago. Such a cavity, when free exit is secured, can be washed out with solutions of germicides of suitable strength.

CATARRH OF THE BRONCHIAL TUBES—ACUTE BRONCHITIS—CAPILLARY BRONCHITIS.

Definition.—The term *bronchitis* is limited to a catarrhal inflammation involving the bronchial tubes, of a caliber above the terminal tubes. Catarrhal inflammation of these terminal tubes, or bronchioles, is designated *capillary bronchitis*, and if associated with atelectasis and catarrhal inflammation of the alveoli, is then known as *catarrhal pneumonia* or *broncho-pneumonia*. If the trachea is at the same time affected with the bronchial tubes, the disease is named *tracheo-bronchitis*. If the inflammation is general over the whole tube, it is called *diffuse bronchitis ;* if limited to a part, *circumscribed bronchitis*. According to the rate of progress, it is *acute* or *chronic*, but the difference is slight.

Causes.—Bronchitis is very dependent on climatic conditions. A humid, changeable, and cold climate favors it, while dryness, uniformity, and warmth of climate have the opposite effect. More than any other single factor does humidity influence and promote the occurrence of bronchitis. Those seasons of the year characterized by the most rapid alternations of temperature, by cold and damp winds, and by excess of humidity, are especially liable to produce bronchitis. All depressing hygienic influences, unsuitable clothing, exposure to damp, cold air—especially when the body is warm and perspiring—are influential factors. In a lowered state of the general health from any cause, the bronchial mucous membrane is more susceptible to evil influences. Bronchitis occurs in greater ratio in men, because they are more exposed to the conditions producing it. Age has an unquestionable influence. The extremes of life are more susceptible, but in infancy bronchitis is more frequent than in old age, but from different causes. The inhalation of irritating gases and vapors and the dust of various occupations will excite inflammation and catarrh. Among the causes must be placed minute organisms, the pollen of plants, which excite local irritation of the respiratory tract, and epidemics of catarrhal diseases. Valvular affections of the heart, which maintain congestion of the lungs and bronchi, necessarily induce a catarrhal state of the bronchial mucous membrane.

Pathological Anatomy.—The initial factor in inflammation of the bronchial mucous membrane is hyperæmia, or increased blood-supply, the whole surface marked by a fine arborescent or punctiform redness, or spots or limited areas only are thus affected. The depth of color depends on the period and intensity of the disease—recent and severe inflammation causing deep redness, and passive inflammation a dark-red, even purplish injection. It is hardly ever the case that the entire bronchial tract is invaded by the redness, but portions of the trachea, a considerable part of the primary and some portions of the second and third divisions of the bronchi. In old cases the redness disappears and is replaced by a grayish, ashy hue, with relatively numerous enlarged and tortuous vessels showing through. Nutritive changes in the epithelium, overgrowth of the glands, and proliferation of the connective-tissue cells of the submucosa, increase the thickness of the mucous membrane. The cartilaginous rings also undergo important changes, and the peribronchial connective tissue is the seat of an active hyperplasia. The new connective-tissue elements displace the cartilage. The secretion of the mucous membrane is changed in character; at first the sudden hyperæmia suspends the production of mucus and the membrane is dry; the next step consists in an increased production of mucus, soon followed by purulent elements, which rapidly preponderate, giving the expectoration a yellowish color. The amount of secretion varies in different cases: when it is deficient, the case is known as *dry catarrh;* when pus is copiously discharged, it receives the name of *bronchorrhœa.* The extension of bronchitis to the alveoli of the lungs and the collapse of lobules constitute catarrhal pneumonia. Emphysema may also result, especially the vicarious emphysema, and when the atelectatic condition happens to many lobules. The bronchial glands frequently participate in the inflammation, become hyperæmic, swollen, and filled with secretion, or the gland elements undergo hyperplasia and ultimately the cheesy transformation.

Symptoms.—There may be catarrh of the upper air-passages, and at the same time there is experienced a raw and sore sensation under the sternum, and a dry, harsh, and rather ringing cough, which awakens pain, and has often a suppressed character because of the pain. At first the cough is dry, corresponding to the dry stage of the mucous inflammation, and is most troublesome in the evening. There are also much muscular soreness and a sense of fatigue, but no other symptoms of illness. In other cases there may be some feverishness, headache, and anorexia. The cough, which was dry, now brings up some mucus, at first only after repeated coughing, but in a short time easily and abundantly, and the expectoration at last has an entirely purulent character, and comes up in globular masses. The fever now disappears, the pain and soreness cease, the cough is easy and less frequent, the appetite is restored, and the return to health is completed in a few days. Such is the course of a simple acute bronchitis (a cold on the chest), which terminates in recovery in about sixteen days. In such a case the changes in the mucous membrane, we may suppose, consist in hyperæmia and swelling, with increased secretion of the glands and more or less destruction of the epithelium. The more severe cases of bronchitis come on with muscular soreness, headache, chilliness, and fever. There is not a single violent chill marking the onset of the disease, but a succession of chills in which there is merely some chilliness felt several times during the course of the day, and having no influence on the fever, which has an exacerbation in the evening and a remission in the morning, or a complete intermission. Sometimes the febrile movement exists without there being any other symptoms for several days, but the more usual onset is the simultaneous appearance of chest symptoms. There is a sensation of heat and stuffing under the sternum; cough, which is accompanied by soreness within the chest, now comes on, and it is dry, harsh, ringing. The frequency and force of the coughing make the diaphragm and chest-muscles sore, and now and then the stomach is emptied in a violent paroxysm. In a few days —usually from three to five—the dryness of the mucous membrane ceases, and abundant secretion of mucus now takes place, and there is brought up frothy mucus, which day by day assumes more of a purulent character. The fever now declines somewhat, but frequently a gastro-intestinal catarrh is lighted up and diarrhœa supervenes. This is apt to be the case with children, in whom the nausea, vomiting, and diarrhœa assume an important position. The coincident development of bronchial and gastro-intestinal catarrh produces a complexus of symptoms to which the term *catarrhal fever* has been applied. In bronchitis the sonority of the chest is not altered from the normal.

During the dry stage the swelling of the mucous membrane narrows somewhat the lumen of the bronchial tubes, but there is no secretion to produce a new sound. The passage of air through narrowed tubes modifies the vibrations, and hence the terms sibilant and sonorous *râles*, audible at this stage, both with inspiration and expiration. When secretion of mucus, muco-pus, and pus succeeds to the dryness, the *râles* are said to be *moist*. Those are *sub-crepitant* which are produced in the smaller tubes, and *mucous* and *sub-mucous* formed in the larger tubes. The largest sounds, or *gurgling*, are produced only in cavities, or that which is equivalent, dilated bronchi. The sub-crepitant is more distinct in inspiration, but all of these *râles* are audible both in inspiration and expiration. Moist sounds are modified by coughing and expectoration—may, indeed, be caused to disappear by them.

The usual termination of these cases of bronchitis is in resolution. The fever ceases, the tongue cleans, the appetite improves, the cough subsides, the expectoration is copious, easy, and purulent, but the amount declines rapidly. Certain types of subjects manifest a great susceptibility to attacks of bronchial catarrh, and the effects do not cease. This is the case in the dyscrasiæ, and when the catarrh is due to cardiac disease there can only be a temporary subsidence in the severity of the symptoms. In those debilitated by constitutional causes, or in subjects of the strumous type, the acute attack passes into the *chronic* form. Acute bronchitis, by an extension of the inflammation to the finest tubes, becomes *capillary bronchitis.* This is often the case in whooping-cough, and in the eruptive fevers—notably in measles. In those debilitated by previous illness, in the old, and in infants, capillary bronchitis is a most serious malady. A sudden increase in the temperature and a marked difficulty of breathing announce the onset of this disease when it arises as just indicated. So difficult is the breathing that the patient calls into use the auxiliary muscles of respiration; unable to lie down, he sits, inclined forward, the arms resting on some support, struggling to get breath, and the respirations, shallow and incomplete, reaching in an adult to forty, in infants to eighty per minute. The difficulty of breathing is incessant; although, now and then dislodging some mucus by coughing or vomiting, there is a temporary alleviation of the distress. At first the respirations, although hurried and oppressed, are normal; but, when the air can not enter, the lungs are not expanded, and the diaphragm is not depressed, the inferior part of the chest and the epigastrium are drawn in with each inspiration instead of being elevated, while the upper portion of the chest remains immovable. At first the face is red, the eye bright, and the skin hot with the unwonted effort, but as the air fails to reach the lungs the blood is not oxygenated, the face becomes pale, the veins enlarged, and the countenance has an increasing duskiness from the accumulation of carbonic acid in the blood. The restlessness and anxiety yield to an increasing stupor, and the approaching cardiac failure is announced by rapidity and feebleness of the pulse. When no efforts succeed in removing the obstruction to the entrance of air, death takes place in four or five days, but the duration is longer if by vomiting or other means the access of air is secured, even for a brief period, to the alveoli of the lungs. When a favorable termination is about to take place, the dyspnœa becomes less urgent, the pulse improves in volume and lessens in rate, the fever diminishes, the expectoration is less viscid and comes up more abundantly, and ten or twelve days from the onset convalescence is fairly inaugurated. More or less simple bronchitis may persist for weeks longer. The physical signs are similar to those of bronchitis, except the differences due to the volume of the tubes attacked. Besides the coarser sounds of bronchitis, the dominating *râle* is the sub-crepitant, audible all over the chest. As in capillary bronchitis collapse of lobules takes place, the physical signs of atelectasis are superadded. These have already been sufficiently discussed.

Course, Duration, and Termination.—Simple bronchitis usually terminates in resolution in about ten to fifteen days. In children the course may be more protracted, and the symptoms more severe, if complicated by gastro-intestinal troubles. The termination may be in the chronic form of the disease. There may be an extension of the morbid action from the larger to the finest bronchial tubes. Capillary bronchitis pursues a more rapid course, and may terminate in four or five days, but it usually continues up to the ninth, even twelfth day. The mortality from capillary bronchitis is large, because of the occurrence of atelectasis and broncho-pneumonia or catarrhal pneumonia.

Diagnosis.—Acute bronchitis is to be differentiated from catarrhal pneumonia and croupous pneumonia. Bronchitis pursues a much milder course, is of shorter duration, and is greatly less dangerous to life. While the moist sounds are the same in the two diseases, the sub-crepitant *râle* preponderates in catarrhal pneumonia, and in the latter the vesicular murmur is replaced by blowing or bronchial breathing and bronchial voice. Bronchitis commences by chilliness persisting for several days—pneumonia by a distinct and severe rigor; in bronchitis there is fever of moderate height—in pneumonia, the range of temperature is very high; in bronchitis, the fever declines gradually—in pneumonia, there is a sudden defervescence; in bronchitis, the sputa consist of muco-pus and pus—in pneumonia, of a peculiar viscid material stained with blood; in bronchitis, there are moist sounds, with sub-crepitant *râle*—in pneumonia, there is crepitant *râle*; in bronchitis, there are no sounds indicating pulmonary lesions—in pneumonia, there are bronchial breathing, bronchial voice, etc. Bronchitis of the larger is to be distinguished from bronchitis of the smaller tubes, by the dyspnœa, by the fineness of the sounds, and the greater danger to life. The onset of catarrhal pneumonia from bronchitis is announced by the increased difficulty of breathing, the rise of temperature, and the diminishing sonority of the chest over the affected parts, with the auscultatory phenomena of consolidation.

Treatment.—The simplest means suffice for an uncomplicated case of acute bronchial catarrh. The combination of tartar emetic (gr. $\frac{1}{16}$) and morphine (gr. $\frac{1}{12}$) in some sirup of lactucarium, or in water, a mustard-plaster to the chest, and confinement to bed, will afford satisfactory relief. In children, sirup of ipecac, sirup of tolu, and paregoric usually suffice. If there is much fever, and the pulse active, tincture of aconite-root (gt. j) should be added to the ipecac and paregoric. When the acute symptoms have subsided, the stimulant expectorants should be used—acetum scillæ, sirup of senega, and sirup of tolu, for example. When the bronchitis is severe, there is high fever, and the inflammation seems disposed to invade the finer tubes, and especially if the finer tubes are invaded, tartar emetic in sufficient quantity to produce a little nausea, morphine in very small doses, and the tincture of aconite, are highly serviceable. The more the finer tubes are invaded, the greater the need of ammonia, carbonate or chloride, and the iodide. Should there be much obstruction, emetics of subsulphate of mercury or of apomorphine must be employed to tide over the emergency, and then the iodide and carbonate of ammonia, in small doses, should be given frequently. Should the temperature rise high and continue so, antipyretics, as cold baths and quinine, more especially the latter, must be administered. A temperature requiring antipyretics may be attained when a simple bronchitis becomes a capillary bronchitis or broncho-pneumonia. A persistently high temperature greatly increases the danger of cardiac failure. If there be indications of such failure, ammonia carbonate and alcoholic stimulants must be freely but judiciously administered. The diminution in the supply of oxygen and the accumulation of carbonic acid are important sources of danger in capillary bronchitis. The timely use of emetics, by giving at least temporary admission of air, will postpone the period of stupor from carbonic-acid narcosis. When bronchitis in children assumes the aspect of catarrhal fever, the remedies employed must be different in character. Nauseants, emetics, and irritants must be discontinued if they have been used. Paregoric, with some carbonate of ammonia, in sirup of tolu, is a good prescription in these cases. In all cases of the different forms of acute catarrh of the bronchial tubes, alimentation is important, but especially so in those cases accompanied by gastro-intestinal disorder.

CHRONIC BRONCHITIS—CHRONIC BRONCHIAL CATARRH.

Definition.—By this term is meant an inflammation beginning in the mucous membrane of the bronchial tubes, chronic in type, and involving not only the mucous membrane, but the substance of the tubes and the peribronchial connective tissue.

Causes.—Chronic bronchitis but rarely succeeds to a pronounced acute attack. Usually the early symptoms escape recognition, or the chronic form is a resultant of not one but numerous acute attacks. This malady is always associated with obstructive lesions of the heart or lungs. It accompanies or is a local development of the dyscrasiæ, as rickets, scrofula, Bright's disease, and of the infectious diseases. The tendency to it may be inherited, or rather a type of mucous membrane disposed to such changes may be transmitted.

Pathological Anatomy.—The mucous membrane is brownish in color, or has a steel-gray color. In other examples, owing to the development of vascular loops, it has a bright-red color. The follicles of the mucous membrane are swollen and enlarged by hypertrophic thickening of the connective tissue, and by accumulation of their contents. The connective tissue, especially of the posterior part of the tubes, and the peribronchial connective tissue, become greatly thickened; the cartilages are invaded and much weakened. Under the strain of coughing, especially if there be at the same time firm pleuritic adhesions, the bronchi yield and dilate. The dilatations are cylindrical, fusiform, and sacculated. In cylindrical dilatations the tube or tubes are uniformly enlarged throughout; in the fusiform variety the enlargement has a spindle-shape, and in the sacculated there is a lateral protrusion forming a sac or a cavity. To these might also be added the moniliform, in which there is an enlargement of one part, then the tube is normal, then again an enlargement, so that the normal portions by comparison with the dilated seem to be contracted.

The secretions in chronic bronchitis differ greatly from the normal. Fragments of the detached epithelium, mucus, and pus-corpuscles, are the morphotic elements, the purulent being very largely in excess. Usually the secretion is very abundant, greenish-yellow in color, and sometimes fetid. When the secretion consists of young cells and mucus corpuscles and granules, it is called *mucous catarrh;* when the cellular elements are not present, and the secretion is viscid, colorless, without odor, and resembling white of egg, it is called *pituitous catarrh* or *bronchorrhœa;* if the secretion is scanty, tough, rather glistening, semi-transparent, and occurs in defined, globular masses, it is entitled *dry catarrh.* Whenever the secretion is retained and undergoes decomposition, as is apt to be the case when the tubes are dilated, especially in the saccular form, it is known as *fetid bronchitis,* the fetor being chiefly due to the fat acids.

Symptoms.—If there be no complications, chronic bronchitis is not attended by fever. When it occurs with disease of the heart, Bright's disease, or other dyscrasiæ, the clinical features are those of the original malady, bronchitis being one only of the morbid complexus. As a substantive affection succeeding to acute attacks, it is slow of development. There are observed, for some years, autumnal and winter seizures of bronchitis, which cease with the warmer and more stable weather of the summer. It may be a number of years before the bronchitis becomes constant, which indicates the existence of permanent changes in the tubes. In the so-called *dry catarrh* there is but little expectoration, and that is brought up with difficulty, and after repeated and most distressing paroxysms of coughing. Next to coughing the most important symptom is dyspnœa, due to the viscidity of the exudation, to the swelling of the mucous membrane, and the implication of the finer tubes. The difficulty of breathing is not considerable when at rest, but exertion at once develops it, and it is accompanied by more or less wheezing. Owing to the impaired elasticity of the lung and the dilatation of the tubes, the upper part of the thorax is kept in the position of maximum inspiration, and the expiration is prolonged and difficult. The result is, that the supply of oxygen is insufficient for the depuration of the blood, and cyanosis appears, the face becomes congested, the lips and mucous membrane bluish, and the superficial veins enlarged. The pulmonary circulation is hindered by reason of these conditions, venous stasis ensues, and œdema slowly develops about the ankles. The habitual difficulty of breathing is now and then varied by attacks which have an asthmatic character, excited by the inhalation of dust, remaining in a crowded apartment, taking cold, and especially by an attack of acute bronchitis with profuse secretion (humid asthma). These seizures are not very protracted, and terminate after some hours by an abundant discharge of mucus. The cases of chronic bronchitis characterized by profuse expectoration differ from the preceding type in several respects—in a more abundant expectoration, in a less troublesome cough, and in less habitual difficulty of breathing. In these cases of so-called *humid bronchitis* there are occasional paroxysms of dyspnœa, due to extension of the morbid process to the smaller tubes, causing difficulty of breathing by swelling of the mucous membrane, by accumulation of secretion, etc. With or without such paroxysms, the chief troubles arise from the cough, which is most annoying at night or in the early morning, and an abundant expectoration. The sputa consist of muco-pus, or of a semi-transparent, albuminous, viscid fluid (bronchorrhœa), or of a greenish-yellow pus, and the variations represent differences in the local changes already designated. Percussion reveals no change in the normal sonority of the lungs in uncomplicated cases. If emphysema, or broncho-pneumonia, or fibroid phthisis have occurred, there will be changes in sonority, but these diseases are not in question. In dry bronchitis, on auscultation sibilant and sonorous *râles* of every variety will be heard ; in humid bronchitis, mucous and sub-mucous, and subcrepitant *râles* will be abundant according to the amount of secretion volving not only the mucous membrane, but the substance of the tubes and the peribronchial connective tissue.

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Course, Duration, and Termination.—Chronic bronchitis pursues an essentially chronic course, but it is diversified by variations in the intensity of the symptoms, by remissions and intermissions. These intermissions are only possible in the early period; after a time the symptoms persist. Chronic bronchitis may continue during a lifetime, and death be caused by some other disease. Recovery may ensue in the milder cases, and is more likely to occur in young than in old subjects. Severe cases of bronchitis lead to the production of other maladies. The long-existing purulent exudation in the tubes, interstitial pneumonia having been produced by the extension of the peribronchial connective-tissue inflammation, excites tubercular deposition. Fibroid phthisis is usually, probably always, produced in this way, chronic bronchitis initiating the series of morbid changes. Emphysema is a result of dry catarrh, for in this case the chronic inflammation is seated in the finer bronchi, the secretion is highly viscid, the membrane much swollen—conditions most favorable to collapse of lobules and emphysema. Hypertrophy and dilatation of the right cavity, venous stasis, and general œdema are also results of chronic bronchitis, and in this way a considerable proportion terminate. The disturbed circulation in the lungs and the venous stasis cause congestion of the liver and of the kidneys, and death may be due to the maladies thus created.

Diagnosis.—The same considerations govern the diagnosis of chronic as of acute bronchitis. The disease with which chronic bronchitis is most apt to be confounded is phthisis. The difficulty of separating chronic bronchitis with sacciform dilatation from phthisis with cavities is very great. The differentiation must rest on the history of the cases, the evidence of pulmonary lesions outside of the cavity, to be discovered in phthisis and not in bronchitis, and in examination of the sputa, those of phthisis containing elastic fibrous tissue, etc.

Treatment.—The indications of treatment vary somewhat with the form. In dry bronchitis, full doses of iodide of potassium, or preferably iodide of ammonium (ten to twenty grains), every three hours when the difficulty of breathing is great, are very effective. For the interval between the asthmatic paroxysms, the best results are obtained by a combination of iodide of ammonia and arsenic, with a balsamic expectorant, as eucalyptol, turpentine, copaiba, cubebs, etc. The persistent use of these remedies will often accomplish important results, and will in all cases afford relief, if not cure. When there is profuse expectoration, quinine with atropine, and codeine, to quiet cough, and the balsams, are the most efficient remedies. If the expectoration is fetid, the free internal use of quinine, eucalyptol, and turpentine, is to be commended, and inhalations of the vapor of turpentine and of iodine, or atomization of benzoate of sodium, carbolic or salicylic acid, or thymol, may be practiced. Of these remedies applied by atomization, carbolic acid is most efficient. In all cases of chronic bronchitis with considerable expectoration, much good results from the persistent use of the now well-known phosphate of iron, quinine, and strychnine. The lactophosphate of lime is also highly useful, probably because of the waste of this important material under these circumstances of profuse suppuration. Arsenic is highly useful when the secretion is not abundant, as in dry bronchitis. It may be combined with the iodides, or with the sirup of the lactophosphate of lime. The hypophosphites, as well as the compound phosphates, are useful when there is waste by suppuration. Alcohol has the power to diminish suppuration and to arrest fermentative processes, and is therefore useful in chronic bronchitis. Whisky is the best alcoholic in such cases. It may be taken with cod-liver oil, the two forming a nutrient of much value—a teaspoonful of cod-liver oil and a tablespoonful of whisky after meals. A generous supply of nutritious aliment is, of course, highly necessary.

As taking cold is the principal cause of attacks of catarrh (employing that term to indicate the nature of the influences causing catarrh), it is highly important to avoid this accident by suitable clothing, by good air, and by favorable hygienic surroundings. If a cold should occur, the patient ought to receive at once an efficient dose of quinine and morphine (gr. xv—gr. ss.). As a humid, variable climate, characterized by cold winds and extremes of temperature, is very unfavorable, a change to a mild, equable, and dry climate should be advised.

PSEUDO-MEMBRANOUS OR CROUPOUS BRONCHITIS.

Definition.—*Croupous bronchitis* is an inflammation of the bronchial mucous membrane, characterized by the exudation of a false membrane. It corresponds to croupous enteritis and to laryngeal croup. It may be acute or chronic.

Causes.—The ordinary causes of bronchitis excite this form apparently, but nothing is known of the conditions which give this direction to the products of inflammation. The cases occur usually in youthful subjects, from six to forty[*] years of age, and in those who have been subject to attacks of bronchial catarrh. A depressed state of the body, and possibly an inherited tendency, are also causes. According to Riegel, pulmonary hæmorrhage sometimes precedes, according to Street succeeds to attacks of croupous bronchitis.

Morbid Anatomy.—There are two forms of the croupous process in the bronchial tubes—the *diffused* and the *circumscribed:* the former are so designated because the exudation extends from the trachea through all the divisions of the bronchi; the latter, because confined to certain tubes. The mucous membrane has been found both intensely injected and pale; the epithelium intact, or entirely removed over the whole extent of the surface covered by the exudation. Sometimes ciliated and cylindrical epithelium has been found embraced in the casts; in other cases none has been found. These contradictory observations are due to the fact that the examinations were made at different stages of the disease. Indeed, displacement of the epithelium is not a necessary part of the process of membrane formation. It is most probable that an albuminous solution is poured out, and white corpuscles migrate, the whole consolidating. It may happen that some epithelial cells are embraced, but this is not necessary. The tubular casts form an outline of the tubes in which they were produced. They may be rolled up into a ball, or expelled in fragments, or as a whole. The author has had a case in which a complete cast of one bronchus and all of its subdivisions was expelled entire. The casts differ much in thickness and length. Those coming from the upper tubes are shorter and straighter, and terminate in fine prolongations; those from the lower tubes are longer, and gradually divide into smaller casts. They are not solid usually, at least the larger casts are not, and contain in their interior mucus and air. They have a lamellated structure, and the lamellæ have a concentric arrangement (Riegel)[†]. The casts are elastic and compact, and bear a good deal of strain. They are whitish or yellowish-white in color, and consist of a "hyaline basement substance,"[‡] sometimes fibrillated, as was the case in the author's observation.

Symptoms.—There are two forms—as regards the clinical features—the acute and chronic. The acute attacks begin as an ordinary acute bronchitis, with chilliness, fever, general *malaise*, a troublesome cough, soreness of the chest, and oppression. These symptoms continue for several days, when more formidable troubles are manifested by an increasing dyspnœa, "livid, swollen countenance,"* high fever, rapid pulse, a dry, harsh, and resonant cough, anxiety, and sometimes hæmoptysis. There may be no preliminary symptoms of acute bronchitis merely, but the disease set in at once by severe difficulty of breathing, preceded by a rigor, and accompanied by high fever. At first the expectoration is that of bronchitis, but in a few days the characteristic casts are brought up with a good deal of coughing and straining. There may be then immediate relief afforded, the dyspnœa subsiding and the cough becoming much less severe. In the course of a few hours, or a day or two, there may be a recurrence of the severe dyspnœa and the straining cough, and more casts will then be discharged. More or less hæmorrhage may occur, or masses of bloody mucus may be expectorated. In the chronic form of croupous bronchitis, there is usually a history of chronic bronchial catarrh, or of some form of pulmonary disease. During the course of such disease, acute bronchial symptoms come on, fever, dyspnœa, and a most severe straining cough, cyanosis, anxiety, etc., during which casts of the tubes are expectorated. Then the symptoms subside, and afterward only those symptoms pertaining to the chronic malady are experienced, until there occurs a return of the paroxysms. In some cases, during a long time—a year—there may be discharged every few days casts; in other cases the attacks may occur two or three times a year.† When the attacks happen at longer intervals, the symptoms are apparently more acute and severe.

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[*] Dr. Street's case—a man aged thirty-nine, "American Journal of Medical Sciences," January, 1880, p. 149.

[†] Ziemssen's "Cyclopædia," vol. iv.

[‡] "Report of Cases of Fibrinous Bronchitis," by Dr. Glasgow.

Course, Duration, and Termination.—The acute cases run their course in a few days. The fatal cases may terminate within the first week, as early as the fourth day, and none continue longer than two weeks. About one half of the cases terminate fatally. In the fatal cases the casts either remain *in situ* or are in part discharged, or are reproduced. The cyanosis rapidly deepens, carbonic-acid poisoning supervenes, the dyspnœa augments, and the patient dies asphyxiated. The chronic form pursues a different course. The attacks recur from time to time, during the prolonged existence of a chronic bronchitis, and a fatal result is reached in an acute attack with symptoms of asphyxia, or by the changes belonging to the associated malady. Other cases are connected with phthisis, emphysema, etc., and pursue a similar course, death occurring usually in an acute suffocative attack.

Diagnosis.—Until the characteristic casts have been discharged, it will be impossible to distinguish these attacks from those of capillary bronchitis. As there are no symptoms of laryngeal stenosis, bronchial will be readily separated from laryngeal croup. A careful consideration of the history of the case will prevent this disease being confounded with a foreign body in the air-passages, the symptoms being much the same in both. It is to be distinguished from catarrhal pneumonia by the changes in the sonority of the lungs caused by the latter, but a suspension of judgment will be necessary until the casts are expectorated in those cases of croupous bronchitis occurring in the course of chronic pulmonary affections.

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* "Transactions of the Pathological Society," vol. xi, p. 23.

† Ibid., p. 24.

Prognosis.—Opinions must be expressed with caution in any case of the acute type, as fifty per cent. prove fatal. In chronic cases the prognosis is grave, because in so many of them lesions exist, which must eventually destroy life. The prognosis is favorable, however, in the chronic cases without complications, as recovery takes place in a majority of them. The prognosis is rendered grave by these indications: severe dyspnœa, cyanosis, stupor, high fever, great extent of the surface affected in the lungs, the extremes of age, little vigor of constitution, and bad hygienic surroundings.

Treatment.—As the extreme urgency of the symptoms depends largely on the obstruction by the false membrane preventing the access of air, the first requisite is to dislodge and remove this obstruction. Active emesis is the most effective means for immediate result, and the most efficient emetic is apomorphine, which should be injected hypodermatically. Next to this is the subsulphate of mercury, which acts promptly without producing depression. Tartar emetic is too depressing, but it may be employed in the absence of the other agents. Sulphate of zinc is safe and effective. The repetition of the emetic is determined by the dyspnœa and cyanosis. Softening the false membrane by inhalation of the vapor of water, especially of lime-water, is highly serviceable. Merely disengaging steam in the apartment is useful, but the utility of the application is greatly enhanced by the addition of lime. The domestic method of producing vapor and atomizing lime is an excellent plan. This consists in slaking freshly-burned lime, the patient inhaling the vapor as it arises. Lime-water may be atomized in the ordinary way. Such softening and solvent applications should precede the emetic.

Great good has been accomplished in these cases by the administration of the iodides, with alkalies. The author strongly urges the use of the iodide and carbonate of ammonia, in small doses every hour or two. It is highly important to prevent a recurrence of the seizures. Remedies having a direct effect on the bronchial mucous membrane, because eliminated by it in part, at least, afford the best prospect of relief. These remedies are the iodides, the balsams and oils, as copaiba, turpentine, eucalyptol, etc., which should be perseveringly administered for a long time. The effect of these remedies is aided by arsenic, which should also be given persistently. The complications of croupous bronchitis should be treated in accordance with the requirements of each case.

STENOSIS OF THE TRACHEA AND BRONCHI.

Definition.—By *stenosis* is meant a narrowing or contraction of the trachea or bronchi, produced by obstruction within and by pressure from without.

Causes.—The trachea or the bronchi are narrowed by interior obstructions and by exterior pressure. In the second group are included enlarged thyroid or goitre; swollen lymphatic glands at the hilus of the lungs and the bifurcation of the trachea; aneurism of the arch of the aorta, especially of the concave and posterior arch; tumors, abscesses, etc., of the mediastinum; and cancer of the lung. In the first group are cicatrices, indurations, and adhesions; neoplasms or new formations; inflammation and thickening of the walls, etc.

Symptoms.—So far as the symptoms are concerned, the cause of the obstruction is of little moment. The most obvious symptom of stenosis is difficulty of breathing, but not the kind of difficulty produced by emphysema, capillary bronchitis, etc., which is expiratory, whereas that due to this disorder is inspiratory. When there is great difficulty, all of the accessory muscles of respiration are brought into action to fill the lungs, but expiration is easy and unobstructed. Notwithstanding the strong efforts put forth to fill the lungs, this is not accomplished, and hence more or less rarefaction of the air in the lungs takes place, so that on inspiration, instead of expanding, certain parts of the chest are drawn in, viz., the lower part of the sternum and the inferior ribs. The movements of the larynx are very slight in tracheal and bronchial stenosis, and very free in stenosis of the larynx. A peculiar whistling, wheezing, crowing, or musical note is produced by stenosis, and the sound of expiration is higher in pitch than that of inspiration. If the obstruction is sufficiently high up in the trachea, the vibration in the column of air may be transmitted to the walls of the organ, producing a defined thrill. The voice is weak and muffled, because of the interruption in the passage of air to the vocal cords. The vesicular murmur is also weakened, obscured by the tracheal or bronchial sounds, or absent. This change may exist in one lung only, if a bronchus is obstructed. If the stenosis is in one bronchus only, the movements of the corresponding side of the thorax are lessened; the vesicular murmur is diminished, obscured or abolished, and there are loud whistling, sonorous, and wheezing sounds, with more or less thrill, while the sonority of the corresponding lung is undiminished. The healthy lung having an increased amount of work to do, there is more or less expansion, the movements are also greater, and the diaphragm is pushed down somewhat. A laryngoscopic examination separates laryngeal from tracheal stenosis, and under favorable circumstances indicates the position and character of the latter. The rational symptoms are those of difficulty of breathing and obstruction to the entrance of air. The face is anxious, the alæ of the nose work, the skin is covered with a sweat, and there is constantly present a sense of the need of air. Besides this constant difficulty of breathing, the severity of which depends on the amount of the stenosis, there now and then occur acute exacerbations of dyspnœa, due either to a fresh catarrh, to a sudden increase of the compressing force, but especially to an asthmatic attack. The ordinary rate of difficulty of breathing may continue uniform for a long period ; but toward the end suffocative attacks come on, which are at first separated by considerable intervals of time, but become nearer gradually, and life is ended by them, or by an intercurrent pneumonia.

Course, Duration, and Termination.—The clinical history is usually divided into three stages : the first consists of the disturbance produced by the growth of the obstruction ; the second, the period of difficulty of breathing and the other symptoms due to the completed obstructing cause, which may continue for a long time ; the third, consisting of the final suffocative attacks. The duration is protracted, and can not be expressed in definite numbers. The ultimate termination of a large proportion is death ; many cases may continue for years without apparently interfering with health, but these are exceptional cases. Cerebral symptoms—coma—may appear toward the end. Death may be caused by pneumonia, œdema of the lungs, etc. Sometimes death occurs suddenly without the warning afforded by severe dyspnœa, caused by the rupture of an aneurism, of an abscess, or rarely without any apparent cause.

Treatment.—The therapeutical management is concerned with the cause of the stenosis, and need not, therefore, be considered here.

ASTHMA.

Definition.—This term has been applied to various morbid states, characterized by spasmodic difficulty of breathing, but it should be restricted to an independent, substantive affection occurring paroxysmally, without any morbid alteration of the breathing organs, and consisting in acute dyspnœa, lasting some hours, and terminating in health. It is appropriately divided into the *idiopathic* and *symptomatic*.

Causes.—Various theories of asthma have been proposed. Without occupying space with details, it will suffice to state that asthma is a *neurosis* of the breathing apparatus, and like other neuroses arises from sources of disturbances in the nervous system, central and peripheral. Like other neuroses, the conditions of the nervous system necessary to its development may be inherited. Nothing is more common than the occurrence of this malady in different generations and branches of a family—the author has known of many examples. Asthma alternates with other nervous affections—with hemicrania, epilepsy, and angina pectoris. Asthma also alternates with affections of the skin—with urticaria, for example; and succeeds to eruptions of the skin, of the herpetic kind (Waldenburg). The pressure of enlarged lymphatics on the pneumogastric nerve has excited attacks. Various peripheral irritations induce asthmatic seizures. Evil intelligence, the association of ideas as connected with particular localities, and other moral causes, will excite attacks. Curious examples are related in regard to the influence of local associations: thus attacks occur on one floor of a house, and not another; on one side of a street, and not the other, etc. Distention of the stomach, indigestion, and flatulence, nasal polypi, certain odors, dust of a peculiar kind, pollen of plants, etc., will excite attacks. The mechanism is plain. In the case of intestinal irritation, the end-organs of the pneumogastric are acted on, the impression is communicated to the pneumogastric nucleus, and reflected over the bronchial and pulmonary branches of the vagus. In the case of affections of the nasal mucous membrane, the filaments of the fifth nerve receive the impression, and, as the nucleus of the fifth and of the pneumogastric lie in close juxtaposition, and are intimately associated in function, disturbance in the one is easily and quickly transferred to the other. Of this relation numerous examples exist. Asthma is more common in men than in women: according to Hyde Salter, of one hundred and fifty-three asthmatics tabulated by him, one hundred and two were men, and fifty-one were women. The disproportion is greater in advanced llfe. Asthma is common in childhood and up to middle age, but occurs at all ages. It is rather more common among the well-to-do classes. Surroundings have but little influence, unless a predisposition exists. Change of locality has a remarkable influence on asthma, but the conditions of climate which prove favorable are most diverse. Some do better in the heart of a great city, others on a dry and elevated plateau, others in a humid valley. Mental and moral influences are more potent than mere climatic peculiarities.

Pathogeny.—As asthma is a neurosis, there are no anatomical changes peculiar or essential to it. There are, it is true, morbid states associated with, but are not necessary to it. Bronchial catarrh is often found, also emphysema, but these are sequelæ or results, rather than a part of the disease. During the existence of the asthmatic paroxysm, an intense congestion has been seen on laryngoscopic examination. There are, at present, two dominant theories of the pathogeny of the asthmatic seizures; the *theory of tonic spasm of the diaphragm*, propounded by Wintrich; the *theory of spasm of the bronchial muscles*, which is the oldest theory, but has the support of Salter, Williams, and Trousseau, and is now sustained by the remarkable investigation of

Professor Paul Bert. The new theory of Leyden[*] has attracted attention by its singularity. He finds in the expectoration brownish cells undergoing granular degeneration, between which are colorless, extremely small but pointed, octahedral crystals, some readily visible, others requiring immersion lenses to find them. These crystals have been examined by Salkowski,[†] with the result to show that they must be composed of a material analogous to mucin. Leyden supposes the asthmatic paroxysm to be determined by a reflex spasm of the muscles of the bronchial tubes, induced by the irritation of the terminal filaments of the vagus by these minute crystals. A more recent and the latest theory is that of Weber (Riegel[‡]), which supposes the concurrence of a number of factors in causing asthma, such as bronchial spasm, catarrh of the tubes, tonic spasm of the diaphragm, cardiac lesions, etc., which is, in fact, a combination of the previous theories, and is, probably, the nearest approach to a true hypothesis in that it adopts all the presumed causes.

Symptoms.—The first attack is sudden, but the succeeding attacks are preceded by prodromes, the significance of which presently becomes apparent to the sufferer. These prodromes are usually acute coryza, some bronchial irritation, headache, and general *malaise*; or the preliminary symptoms may be those of indigestion—acidity, pyrosis, flatulence, hiccough, sneezing, etc. The first attack is nocturnal. The victim, after some uneasy sleep, is suddenly aroused by an intense anguish in his chest; he is stuffed up and struggles for air, jumps from the bed and rushes to the window, or he sits up, leaning forward on his arms, and uses all his strength in the effort to get more air. The breathing is accompanied with loud wheezing, the face becomes flushed and at the same time cyanosed, and is bathed in perspiration, the eyes stare, the eyeballs protrude, and the muscles of the neck start prominently up, as they are called on to aid in the effort to get air. The difficulty of breathing soon reaches a point that the inspiration is nothing but a gasp, the lips become pallid, the cyanosis deepens, and it appears to the patient that every minute must be his last. After some minutes or hours the respiration becomes a little easier, more air enters the lungs, the cyanosis subsides, and gradually the paroxysm ceases. Eructations of gas give great relief as the breathing becomes easy, and the bronchial tubes pour out an abundant mucus secretion, the expectoration of which also contributes to the ease of respiration now rapidly increasing. A free urinary discharge also takes place, the urine being pale, and of low specific gravity. The patient, exhausted with the violence of his efforts to get air, sinks into a profound sleep, and is bathed in perspiration. The whole duration of an attack rarely exceeds six hours, and may, indeed, be no more than one hour. On the following day there are experienced muscular soreness, languor, and debility, but all unpleasant feelings subside and disappear in twenty-four hours, and a normal condition is maintained until the next attack. Instead of a single paroxysm there may be only slight remissions, and one attack succeed to another, with exacerbations, so that the patient can not lie down at all, can take but little food, and is, after some days of suffering, utterly exhausted. The attacks are not exclusively nocturnal, but do sometimes occur during the day. A diurnal attack must be the rule in those cases brought on by the inhalation of some kinds of dust, gas, or vapor; as from powdered ipecac, etc. On percussion, the sonority of the thorax is increased in the vertical diameter from one to two inches, and also transversely, and does not change either on inspiration or expiration. The percussion-note is highly resonant all over both lungs, and has somewhat the tympanitic quality. The "bandbox-tone," by which it is described by Bamberger, is eminently characteristic. The vesicular murmur is either absent or greatly enfeebled, or obscured by the loud, wheezing, whistling, sibilant sounds. During expiration the sibilant, sonorous, whistling, cooing, sighing sounds are more pronounced and of longer duration. Toward the close of an attack moist sounds occur. The explanation of the physical signs present in an attack of asthma is afforded in the condition of the chest. The diaphragm is depressed below its ordinary position by tonic contraction; the chest, which assumes a distended, globular shape, is fixed in the position of forced inspiration. The lungs are filled with air, but it is residual air, and is not renewed; and, notwithstanding the effort put forth by the patient, the little air which can be introduced only adds to the distention. Expiration is prolonged, laborious, wheezing, and much more so than inspiration. Spasm of the muscular fibers of the bronchi is perhaps only one element in the obstruction to the expiration of air; tonic contraction of the diaphragm contributes not a little to the result. The fullness of the cephalic veins and the cyanosis and lividity of the face are due to the contraction of the cervical muscles preventing the return of blood, and to deficient oxygenation of the blood. While the face is flushed and the head hot, the feet are cold. The sputa are wanting in the beginning, but appear abundantly at the close of the paroxysm; they are frothy, grayish-white, or reddish-white if mixed with blood, and consist of mucus corpuscles, cylindrical and ciliated epithelium, and peculiar "yellowish-green clumps" in which are imbedded Leyden's crystals.

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[*] Virchow's "Archiv," vol. liv, p. 324, "Zur Kenntniss des Bronchial-Asthma."

[†] Ibid., p. 344.

[‡] Ziemssen's "Cyclopædia," vol. iv.

Course, Duration and Termination. — Asthma is an essentially chronic disease, not incompatible with long life, and with good, even vigorous health, during the intervals between the seizures. The paroxysms last from two to six hours, but sometimes they persist for days. Of itself, asthma is never fatal to life, but changes in the organism are gradually effected by the disturbance in the respiratory function, which may cause death. Emphysema, dilated right cavities, dropsy, or cerebral hæmorrhage, may be brought on by the long-continued operation of the cause. Much depends on the number of the paroxysms. There may be very few or very many. They may be mild at first, and become more severe, or they may commence and persist with the greatest severity. They may disappear suddenly, and never occur again. According to the behavior of the disease will vary the sequelæ. Asthma may also occur as a complication of some existing disease—as, for example, emphysema, chronic bronchitis, etc.

Diagnosis.—It is not possible to mistake asthma when the history is known. The first attack may be confounded with œdema of the glottis or spasm, paralysis of the vocal cords, and stenosis of the trachea. Laryngoscopic examination may serve to differentiate at once, by recognition of the lesion. The most important means of determining, besides the history and the direct exploration of the larynx and trachea, is the character of the dyspnœa. In laryngeal or tracheal obstruction, the dyspnœa is *inspiratory*, in asthma it is *expiratory*. In œdema of the glottis, while inspiration is difficult, expiration is easy and unobstructed; with inspiration there is a loud sibilant or crowing noise, and expiration is silent.

Treatment.—To relieve the paroxysm is the most pressing duty. There is no medication so effective as the hypodermatic injection of morphine (from $\frac{1}{12}$ gr. to $\frac{1}{4}$ gr.). An efficient dose of chloral hydrate is often equally effective (℈j—ʒ ss.). As soon as the patient comes under the influence of either remedy, the difficulty of breathing begins to subside. The best results are obtained from a combination of the two remedies—morphine hypodermatically and chloral by the stomach—but in smaller quantity than when administered separately. Nitrite of amyl (by inhalation, three to five minims) sometimes affords relief, but nitro-glycerin solution by the stomach is more effective, and is a highly useful remedy. In many cases iodide of potassium or sodium, in full doses, will arrest the paroxysms very remarkably. From fifteen to twenty grains, every two, three, or four hours, are usually required. It is better practice to give iodide with bromide of potassium, and to each dose of the solution may also be added a drop or two of Fowler's solution of arsenic. This combination is to be commended, especially in the cases which persist for some days. Much relief is afforded by fumes of stramonium and other narcotics; old asthmatics often depend on fumigation to the exclusion of all other remedies. Pastils, or cigarettes containing leaves of belladonna, stramonium, tobacco, grindelia, and poppy, in equal portions, steeped in a saturated solution of nitre and dried, are ignited and the fumes inhaled. Iodide of ethyl inhaled is effective, for to the calmative action of ethyl is added the influence of an iodide. There are proprietary pastils sold, but, under what name soever they appear, the composition, with unimportant differences, is about as stated above. Belladonna-leaves saturated with nitre afford as good results, usually, as the more complicated pastils. Simple nitre-paper gives ease for a time. The new California remedy, *grindelia robusta*, has undoubtedly great power to arrest a paroxysm of asthma. Three to five grains of the extract or the fluid extract (ʒ ss.) can be given every hour or two. Grindelia is often useful as a fumigant. The debility caused by asthmatic paroxysms is best removed by quinine and iron, the former in considerable doses. This practice is especially to be commended when the paroxysms recur frequently. To prevent a return of the attacks, arsenic is very useful, and is most effective in combination with the iodides. In debilitated subjects, quinine, arsenic, and belladonna may be given steadily for some weeks or months, as the case may be. Asthma, like other neuroses, is capricious in its behavior toward remedies. The remedy succeeding at one time may fail utterly at another time, so that the treatment must be varied accordingly. Hence it is necessary to be fertile of resources in the treatment of this disease. Besides the methods of treatment already mentioned which are most approved, there are others less desirable which should receive some notice. Nauseants, as ipecac, tartar emetic, and lobelia, afford relief by inducing relaxation consequent on the nausea. When there is much catarrh, or the attack of asthma is due to an acute catarrh, good results are obtained by small doses of tartar emetic ($\frac{1}{16}$ gr.) with morphine ($\frac{1}{12}$). A few drops of wine of ipecac (five to ten) every five minutes, until some nausea is experienced, may lessen the oppression remarkably. During the paroxysm, nauseant doses of lobelia (♏ xv—ʒ ss. of the fluid extract) are very effective in stopping the dyspnœa. Recently quebracho has been brought forward as a remedy for dyspnœa, which it often surprisingly relieves. From twenty minims to ʒ j of the fluid extract may be given every hour or two until relief is had.

The application of ammonia to the posterior wall of the pharynx is practiced by the French, but this practice is strongly condemned by Jaccoud. He, however, permits the application of ammonia by impregnating the air of the apartment. The inhalation of oxygen and of compressed air relieves the breathing somewhat, but ether and chloroform are much more effective. Ethyl bromide has lately proved remarkably effective by inhalation.

In the treatment of asthma there is no point of greater importance than careful regulation of the diet. Hyde Salter much insists on this, and the author has had abundant confirmatory observation. The diet should be light and easily digestible, and as little bulky as possible. It should consist, therefore, chiefly of animal food, and to this may be added a little fruit and a few of the succulent vegetables, but starchy and saccharine substances and milk should be excluded. In this prohibition bread is included, as it is particularly apt to disagree. Articles of diet that are fried, pastry, cakes, and sirup, etc., are highly objectionable. Meats should be broiled or roasted. Boiled meats and soups are improper. There should be as little fluid drunk at meals as possible, but a little black coffee may be allowed at breakfast.

DISEASES OF THE LARYNX—ACUTE CATARRH OF THE LARYNX—LARYNGITIS.

Definition.—By *acute catarrh of the larynx* is intended an inflammation involving the mucous membrane—a catarrhal inflammation. There is also a chronic form of the disease—chronic inflammation.

Causes.—The mucous membrane of the larynx is in a position to be quickly and easily affected by external agents of a gaseous or aëriform kind—such as ammoniacal gas, chlorine, tobacco-fumes, etc. Very fine solid particles may be carried in the air in sufficient quantity to excite an irritation of the laryngeal mucous membrane. But the organ is more frequently affected by the condition of the atmosphere itself. The long-continued inspiration of air contaminated by respiration is very apt indeed to cause congestion of the mucous membrane, especially when to this is added the sudden contact of cold air. Too prolonged exertion of the voice may also excite a catarrhal inflammation, especially when the exertion is made in the open air. "Taking cold" is a fruitful cause of laryngitis. There may be an extension of trouble from the pharynx and from the face (erysipelas). Influenza may extend to the mucous membrane of the larynx. Inflammation of the larynx is not an infrequent complication in the course of the infectious diseases. Climate has an unquestionable influence ; humid, cold, and variable climates increase the disposition to affections of the larynx, while warm and equable climates lessen the tendency to these diseases. Affections of the larynx occur at all ages, and both sexes are equally liable in proportion to their exposure to the causes.

Pathological Anatomy.—In the mildest cases there is a transient hyperæmia of the mucous membrane—in certain situations—over the arytenoid cartilages, the ventricular bands, the posterior ends of the vocal cords, and the space between the arytenoid cartilages. In more severe cases there is a good deal of swelling as well as injection of the ventricular bands, the epiglottis, the ary-epiglottidean folds, and the inter-arytenoid space, etc. The color in severe cases, instead of being reddish, is a dark, reddish-brown.

Symptoms.—In the mildest cases there is no constitutional disturbance. The local symptoms consist in heat, rawness, and tickling, referred to the larynx and pharynx. When the thyroid cartilages are pressed, unusual soreness, irritation, and severe pain are experienced. There are also present dryness, and a feeling of a foreign body stick-ing in the throat. Swallowing causes pain by the upward movement of the larynx, and by the pressure of the bolus on the larynx as it descends to the stomach. In the more severe cases the onset of the disease is announced by some chilliness and general *malaise*, followed by moderate fever, anorexia, etc., for several days. Cough occurs at once, and it is noisy, harsh, hoarse, or toneless; or, in children especially, has a ringing, sonorous, so-called "croupy" character. The cough is dry, and produces a sensation in the larynx as of scratching over a raw surface; but in a short time secretion is poured out, and then the cough has a loose character. At first some frothy mucus is expectorated; it may be streaked with blood occasionally, but in the rare hæmorrhagic form pure blood may be expectorated. The sputa soon assume the appearance of muco-pus, the pus elements predominating; and it contains also cast-off ciliated epithelium, young cells, etc. At first the voice is thick, and becomes hoarse on talking; but as the case progresses the hoarseness deepens, and at length there is aphonia. Dyspnœa rarely occurs to adults in simple mucous laryngitis, but in children spasm of the glottis may come on, when there is extreme dyspnœa in brief paroxysms. But, as this disorder will be discussed in a separate section, its consideration as a symptom of laryngitis is postponed. A sense of oppression and need of air is caused if there be much swelling of the vocal cords or ventricular bands in the case of adults—a condition of things not apt to occur unless there be some effusion into the sub-mucous connective tissue. Besides hoarseness, which may end in aphonia, there may be various alterations in the tone of the voice, high pitch or low pitch, and its timbre may be subjected to corresponding variations. The peculiarities of voice are due to swelling of the mucous membrane, variations in tension of the vocal cords, and the condition of the muscles moving the arytenoid cartilages. The tone of voice is hoarse and rough from swelling of the cords, discordant from the difference in the rate of vibrations of the two cords, high-pitched if the tension in the cords is great, low-pitched if the tension is low; or there is a double tone, now high, now low, if the cords vibrate with opposite tension. On laryngoscopic examination the state of the mucous membrane, of the vocal cords, ventricular bands, etc., can be made out, and the changes described verified.

Course, Duration, and Termination.—Acute laryngitis passes through its course in a week, if mild; but the more severe cases may occupy three weeks to a month. Mild as well as severe cases may continue indefinitely by repeated relapses, and at last assume the chronic form. Under some circumstances a simple laryngitis may assume formidable proportions by the extension to the sub-mucous connective tissue.

Treatment.—Confinement to bed for the more severe cases, and to a uniformly but not too highly warmed apartment for the milder cases, is essential. The air of the apartment should be kept moist by the vapor of water disengaged in it. For the relief of the inflamed mucous membrane, tincture of aconite-root—one drop for a child and two drops for an adult every two hours—is highly efficient. If there be much cough, and especially if the cough have the "croupy" character, two to five drops of the deodorized tincture of opium and one or two drops of fluid extract of ipecac may be given together. Application by spray douche of a solution of morphia to the throat is an excellent means of relieving cough, but is not so generally available as the internal administration. A very minute quantity of tartar emetic, with paregoric and sirup of lactucarium, is also an efficient combination. A hot or cold pack should be wrapped about the throat after a brief application of mustard; and, if the case is just beginning, the feet should be placed in a mustard foot-bath. If there be a tendency to spasm of the glottis, bromides should be used. Bromide of potassium may be given with any of the combinations above mentioned.

Prophylaxis is very important in the case of those who have frequent attacks, especially if a phthisical tendency exists. They should wear flannels and protect the feet against dampness, while at the same time they should avoid warm wrappings, especially furs about the throat. The tendency to take cold may be obviated by a daily morning cold sponge-bath, and by keeping up the general health. During a variable season, taking cold may be prevented by the daily morning administration of five to ten grains of quinine, and the access of an impending attack may be prevented by a full dose of quinine and morphine (15 grs.—gr. ¼–½).

CHRONIC LARYNGITIS—CHRONIC CATARRH OF THE LARYNX.

Definition.—*Chronic laryngitis* is an inflammation of the mucous membrane, less active in type than, but the same in mode as, the acute inflammation.

Causes.—The chronic form of catarrhal inflammation of the larynx arises under the same conditions as the acute form, or it succeeds to an acute, or is a result of repeated acute inflammation. Tobacco-smoking, spirit-drinking, and careless use of the vocal organs in speaking, reading aloud, or singing, are all influential causes, the most important, in fact, in our day. The middle period of life and the male sex are predisposing causes.

Pathological Anatomy.—The changes described as occurring in the acute form are the initial lesions in the chronic, except that in the latter the color is deeper red or brownish, the mucosa is more swollen, and the submucosa as well as the mucosa is thickened and indurated. Swelling of the inter-arytenoid fold of mucous membrane and of the ventricular bands (false vocal cords) occurs to the degree that the movements of the arytenoid cartilages are interfered with, and consequently of the vocal cords also. The epiglottis is likewise swollen and thickened, and marked by enlarged and varicose veins. The vocal cords themselves are injected, and their margins roughened. The follicles of the mucous membrane are enlarged by accumulation of their contents in part, but much more by hyperplasia of the surrounding connective tissue. The enlarged follicles or glands, more or less thickly distributed over the surface, give to the mucous membrane a granular appearance. Very rarely hyperplasia of the connective tissue underlying the vocal cords takes place ; the new tissue contracts, and deformity, with stenosis, is the ultimate result.

Symptoms.—Various uneasy sensations are felt in the larynx—a sense of heat, and an irritation compounded of itching and scratching of a tender surface ; this leads to hawking and clearing the throat as if some obstruction were present. Exposure to cold air increases these sensations, but still more irritating is prolonged talking, especially in the open air, leading to frequent swallowing of saliva. The voice is husky, and becomes so much so by talking that frequent efforts to clear the throat are necessary. The voice becomes hoarse, rasping, and deep, or it is high-pitched, and unexpectedly drops into falsetto. As much effort is necessary to get out the sounds, these patients acquire a straining tone and manner, and now and then, amid husky and hoarse, almost toneless sounds, they utter a more distinct and intelligible sound, giving an eccentric and variegated expression to the conversation. The effort required makes talking very fatiguing. In the morning the most severe paroxysms of coughing and straining are experienced ; the secretion accumulates during the night, and it is detached with difficulty, so that much coughing, hawking, and straining are necessary. The secretion is in the aggregate not considerable, and consists of a tenacious mucus, with some pus-corpuscles.

Course, Duration, and Termination.—It is a very chronic malady and is subject to exacerbations and remissions. Care in the management of the organ, and of the general health, rest, and appropriate treatment, bring relief, but abuse of the organ, irregularities of life, and the absence of all treatment, will restore the diseased state to full activity. Years may be passed in this way, the general health meanwhile not suffering from the laryngeal disease. Cures may be effected in favorable cases, if proper treatment is carried out faithfully for a sufficient period of time, but the difficulties in the treatment, the self-denial to be practiced, and the duration of the case, should not be concealed from the patient.

Treatment.—Any effective treatment must include local applications, directed by the laryngeal mirror and by spray. As there is a large extent of surface involved, and as the increased blood-supply is the leading pathological factor, the application of medicated spray may be sufficient of itself. A great number of medical agents are so employed—a solution of tannin (gr. v—℥ j), of sulphate or acetate of zinc (gr. j—℥j), of chlorate of potassium (gr. v—℥j), of bromide of potassium (gr. x—℥j), of nitrate of silver, with care (gr. j—℥j), and of morphine sulphate if there is much irritability. Solution of nitrate of silver is applied by the brush directly to the interior of the larynx. Ziemssen recommends in inveterate cases the solid nitrate, which is applied by the caustic-holder directly. Such external applications as the tincture of iodine, the ointment of the red iodide of mercury, etc., are serviceable as counter-irritants. The larynx must be kept at rest as long as practicable. Taking cold, sudden changes of temperature, exposure to draughts, must be avoided. The general health must be maintained by a suitable mode of life. Change from a variable to a more equable, and from a humid and cold to a warm and dry climate, will often have a most favorable effect on the case.

ŒDEMA OF THE GLOTTIS—INFILTRATION OF THE LARYNX.

Definition.—*Œdema of the glottis* means a serous effusion into the sub-mucous connective tissue. The disease or condition intended by this term is an obstruction to breathing produced by an infiltration of the larynx by any kind of fluid.

Causes.—An inflammation of the mucosa may extend to the sub-mucosa, and cause œdema. A deep-seated phlegmon of the neck, or of the tonsil and the base of the tongue, may involve the larynx by the diffusion of the pus under the mucous membrane. An inflammation of the cartilages or of the perichondrium may result in a similar purulent infiltration. Erysipelas of the face, typhoid fever, or scarlatina, may be unexpectedly terminated by a sudden effusion into the sub-mucous connective tissue. During the course of Bright's disease, œdema of the glottis may occur, or this may be the first symptom of the malady to attract attention.

Pathological Anatomy.—The œdema exists in those parts containing the most abundant and loose connective tissue—in the ary-epiglottic folds, the glosso-epiglottic ligament, at the base of the epiglottis, and in the inter-arytenoid space. When the inferior or true vocal cords are inflamed (one or both), the cord changes its color, and instead of appearing white, glistening, and brilliant, is dull, grayish-red, or violet-red, in patches, the vessels enlarged and varicose. When œdema exists without inflammatory changes, the sub-mucous connective tissue of the ventricular bands especially, and of the folds mentioned above, is distended with a serous fluid, and has the translucent appearance of a fish's swimming-bladder. The ventricular bands project forward, almost meeting in the median line, and shutting from view above the vocal cords. The epiglottis sub-mucous tissue may also be distended in the same manner, giving to that organ the same pellucid and semi-transparent appearance. If the swelling be due to purulent infiltration, the epiglottis, the aryteno-epiglottidean folds, and the ventricular bands, will be swollen, and present a deeply congested, reddish-brown or violet tint, with here and there spots of a yellowish hue. A very considerable collection of pus may form when the base of the tongue, or the loose connective tissue beneath the tonsils, and the tissues of the larynx are simultaneously involved. A serous infiltration sufficient to cause fatal œdema has disappeared in the death-agony, or immediately after, leaving but small traces of the mischief to account for the formidable symptoms.

Symptoms.—Infiltration of the larynx, succeeding either to some inflammatory process in the neighborhood or of the larynx itself, or coming on in the course of some constitutional malady, adds its special features to the symptoms of the preëxisting disease. These are a sensation of distress or actual pain in the pharynx and larynx; painful dysphagia; dyspnœa; or paroxysms of a suffocative character. The sensations referable to the larynx consist of constant oppression as if a foreign body were wedged in the organ, and more or less severe soreness and pain shooting through the whole area occupied by the purulent infiltration, if that be the cause of the symptoms. There may be in attempts to swallow only a sense of soreness or of obstruction, but in the case of inflammation and swelling there will be acute pain. The feeling of the presence of a foreign body and the accumulation of saliva incite the act of swallowing, which is the more painful the more frequently it is repeated. When there is extensive infiltration, swallowing may become impossible, and then the saliva is permitted to dribble from the mouth. At first the cough is dry, rather harsh, and somewhat resonant, but as the swelling proceeds it becomes stridulous and suppressed. The peculiar difficulty in inspiration is the most characteristic symptom. At first a slight sense of stuffing of the larynx and huskiness of the voice are experienced, but the sensation of stuffing grows tighter, and the inspiration becomes prolonged and with a very obvious effort. A hissing, stridulous, somewhat snoring noise accompanies the inspiration, but expiration is easy and noiseless. As the inspiration increases in difficulty, all of the muscles needed to expand the chest, and the accessory muscles of inspiration also, are brought into play. The inspiration is difficult, because, in drawing in the air, the swollen mucous folds are brought together in the center, and the more strongly the effort is made the more tightly the folds are approximated—for, the cartilages of the larynx keeping the lower cavity open, where a partial vacuum is created by the expansion of the chest, the incoming air pushes the mobile folds of swollen mucous membrane before it, and hence, the more powerful the attempts at inspiration, the more tightly the folds are wedged into the narrow space. Expiration also becomes difficult when the swollen folds become immovably distended, and fixed in more or less close apposition. When this occurs, expiration becomes stridulous, whistling, crowing, and difficult, but not usually in the same degree as inspiration.

In the more formidable cases, the obstacles to the entrance of air may become extreme in a short time, the patient dying asphyxiated. In many other cases the group of symptoms just mentioned are varied by attacks of suffocative breathing produced by spasm of the muscles of the larynx. Excited by cough, by attempts at swallowing, or the accumulation of secretion, etc., on a sudden the breathing is arrested, the face gets blue, the eyes start from the head, there are wild gasping, a terrified expression, and death seems imminent. Death may occur in such an attack. Consciousness may be lost, and then the breathing may be resumed ; again, in other cases—but usually the paroxysms do not proceed so far as unconsciousness—air enters the lungs, and the ordinary difficulty of breathing goes on as before. The existence of the obstruction can usually be made out by carefully passing the index-finger over the base of the tongue, when the swollen epiglottis and aryteno-epiglottidean folds may be felt. It is generally impracticable to use the laryngeal mirror when the case is well advanced, but, earlier, valuable information may be gained by its use.

Course, Duration, and Termination.—The most acute cases are those occurring during the course of some infectious malady, as typhoid. The effusion takes place in a few hours, and the patient expires in a short time, asphyxiated. Such may be the course in cases of scarlatina also. In the more chronic kinds of laryngeal disease, if œdema occur, the progress of obstruction is slower ; there may be days passed between the first attack of spasmodic dyspnœa and the fatal result from the asphyxia of œdema. The duration of infiltration of the larynx varies from a few hours to several days.

Diagnosis.—From the difficult breathing produced by capillary bronchitis, emphysema, and asthma, that of infiltration of the larynx is distinguished by the important characteristic of *difficulty in inspiration*, whereas in the former the *difficulty is in expiration*. The aid afforded by digital exploration and by the mirror, when practicable, will enable a diagnosis to be made at once. Passing the index-finger carefully over the base of the tongue, the swollen glosso-epiglottic folds, etc., can be felt. Croup, or laryngismus stridulus, foreign bodies, polypi of the larynx, and aneurisms of the aorta involving the recurrent laryngeal nerve, may produce symptoms similar to œdema. The attacks of pseudo-croup come on suddenly, occur at night, are quickly relieved, and between the paroxysms there is no trouble of any kind. The presence of foreign bodies and polypi is determined by the use of the laryngeal mirror, and by the difference in the rational symptoms. The history of the case, the sudden occurrence of suffocative attacks after the accidental inhalation of some foreign body, and the coming on or cessation of difficult breathing according to the position of the object, are characteristics differing from those due to œdema. The symptoms produced by laryngeal polypus are of slow development, but the mirror enables a view to be had of the growth, revealing a condition of the larynx very different from that of œdema.

Treatment.—To open the trachea is necessary if suffocation is imminent, but, before resorting to such a severe measure, scarification of the swollen membrane should be practiced, according to the method of Dr. Gurdon Buck, of New York. A scalpel wrapped, but leaving the point free, is passed over the tongue, guided by the finger, and when the swollen parts are reached the cutting edge is turned against them, and free scarifications are practiced. If pus is reached, a free incision is necessary to evacuate it. In the case of purulent infiltration the act of vomiting may, happily, effect a rupture of the depot. Vomiting, for this purpose, is best induced by the hypodermatic injection of apomorphine, since swallowing becomes so difficult in these cases. When the infiltration is serous, absorption may be effected by the free salivary and cutaneous discharge induced by pilocarpine. From $\frac{1}{12}$ grain to $\frac{1}{6}$ grain should be given subcutaneously, usually twice or three times in twenty-four hours. Inhalation of ethyl iodide should be frequently practiced, and the air of the apartment should be kept moist by the vapor of hot water. If attacks of laryngeal spasm threaten asphyxia, ethyl bromide can be cautiously inhaled with advantage. The free exhibition of iodide of potassium—ten grains every two hours—is much to be commended in cases of inflammatory character. If uræmic in origin, besides the pilocarpus, digitalis infusion should be given to stimulate the renal functions, and compound jalap powder to cause free intestinal discharges. As the effusion is forming, full doses of quinine should be given before the pilocarpus, and subsequently to support the vital powers reduced by the loss of fluid. Quinine, in full doses, is more distinctly serviceable when the infiltrating material is pus. If the onset of the disease is inflammatory, and the effusion into the submucosa is the result, tincture of aconite-root should be freely administered, and quinine should also be given to prevent migration of the white corpuscles. As this disease very rapidly depresses the vital powers, it is important to supply the system with nutritious aliment from the beginning. The careful administration of stimulants is also necessary. If swallowing becomes very difficult and but little aliment enters the stomach, the amount taken should be supplemented by "rectal alimentation." Defibrinated blood should be injected into the rectum, and nutrient enemata should also be employed.

TUBERCULAR ULCERATION OF THE LARYNX—LARYNGEAL PHTHISIS.

Definition.—Ulceration of the laryngeal mucous membrane is a frequent complication of phthisis, and sometimes, indeed, precedes the pulmonary lesions. Although a symptom in consumption, its independent importance seems to require more elaborate treatment.

Causes.—Virchow * suggests laryngeal tuberculosis as the best manifestation for the study of true tubercle. Miliary tubercles deposited superficially readily induce ulceration, but follicular deposits and infiltration of the basement membrane cause more extensive and destructive changes. Hence to primary and secondary tubercular deposits are due the ulcerations of the larynx.

Pathological Anatomy.—Those parts of the larynx affected by tubercular ulcerations are most exposed to injury by the performance of their proper functions in phonation and deglutition. These parts are the mucous membrane of the vocal processes, the vocal cords, inter-arytenoid region, the aryteno-epiglottidean ligaments, and the cartilages of Santorini. The most superficial ulcers are flat, with a grayish base, and often very extensive from a coalescence of smaller ones. These are probably ulcers by corrosion, the ichorous matters from the lung-cavities setting up an infection in a part—the seat of a catarrhal process. Another form consists in tubercular deposition in the follicles, disintegration and loss of substance, the resulting ulcer having the characteristic "grape-shape" (Rindfleisch). As the follicles lie in the sub-mucous connective tissue, it is readily seen how deep must be the ulcers thus produced. Extensive excavations result from the spread of the destruction laterally and the union of several ulcers. These ulcers, forming on the laryngeal cartilages, soon extend to and ultimately involve this structure. Still another variety of ulcer is derived from the disintegration of tubercle granulations deposited in the sub-epithelial layer of the mucous membrane. The extent of injury to the larynx varies greatly. The mucous membrane of the vocal processes, of the posterior wall of the larynx at the base of the arytenoid cartilages, of the vocal cords, and elsewhere, is destroyed. The vocal cord may not only be deeply ulcerated but even loosened from its process (Von Ziemssen). The ulcers penetrating to the cartilages set up perichondritis, necrosis, and disease of the crico-arytenoid articulation. Thus the mucous membrane of the larynx in its entirety may be the seat of ulcerations, the sub-mucous tissue becoming œdematous and the muscles relaxed and fatty.

Symptoms.—The earliest implication of the larynx in disease is announced by changes in the voice, by hoarseness on making any considerable and prolonged effort, or on taking a slight cold. The changes seen on laryngoscopic examination at this stage are either pallor, anæmia, and paresis of the muscles, or hyperæmia, and some swelling of the mucous membrane of the inter-arytenoid region, etc. Catarrh of the mucous membrane soon comes on, and is followed by superficial erosions and flat ulcers in the region first attacked, these solutions of continuity being surrounded by a zone of more or less intense hyperæmia. In the further progress of the cases, deep and extensive ulcerations occur, the vocal cords becoming ragged, and the edges of the ulcers forming irregular excrescences which may and have been mistaken for polypi. The ulcerations extend over the epiglottis and the arytenoids, and extensive losses of substance occur in advanced cases in the former organ. The weakness and ready failure of the voice and the easy occurrence of hoarseness, noted at first, increase in every way as the ulcerations extend ; hoarseness presently becomes habitual and constant, and finally the voice loses its tone, the patient speaking with difficulty in a husky whisper. Paresis of the laryngeal muscles and œdema of the sub-mucous tissue are in part responsible for the failure of phonation. The pain at first is trifling, a mere sense of heat, but, as the cases progress, much soreness and burning are referred to the larynx, and are increased by the attempts at swallowing. Frequent efforts to clear the larynx of the muco-pus and detritus are made, with the effect to increase the soreness and burning pain. The glottis becoming damaged and the aryteno-epiglottidean folds swelling with œdema, as well as damaged by ulceration, the larynx is imperfectly closed in the act of swallowing, and hence, particles of food and drink entering it, paroxysms of violent coughing and suffocative attacks are provoked.

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* "Die krankhaften Geschwülste," Bd. ii, p. 645.

Course, Duration, and Termination.—The progress of these cases is greatly affected, as might be expected, by the extent of the laryngeal disease and of the pulmonary lesions. In some cases of laryngeal phthisis, so called, the progress of the disease is due chiefly to the extent of the mischief in this organ ; in other cases the pulmonary disease is relatively more active. The duration of the laryngeal malady is therefore a question of the condition of the lungs. When, by reason of the changes in the larynx, swallowing becomes difficult, the nutrition suffers because of the failure to obtain sufficient aliment. No exact limits can be set as to the duration of the case. In some cases the progress of the pulmonary and of the laryngeal lesions is equally rapid ; in other cases the lung-disease is slow, and the laryngeal ulcerations are limited to a few points ; in still others the ulcers of the larynx cicatrize, and an apparent arrest of phthisis occurs, to be followed, after some years, by a renewed activity in the morbid process.

Diagnosis.—We have in the laryngoscope an accurate means of determining the extent of the ulcerations, and from them to judge of the condition of the cartilages and articulations. Varicose prolongations of the margins of ulcers may be mistaken for polypi, an example of which error fell under the author's notice, as made by a celebrated specialist. Careful inspection, the head well extended to bring the excavations into view, will enable the observer to get a correct notion of the local condition. More frequently the error made by specialists consists in the failure to properly appreciate the condition of the lungs, and to ascribe the symptoms solely to the laryngeal lesions. As it is an extremely rare, almost an unknown, event to have laryngeal tuberculosis exist alone, the inability to discover the pulmonary disease should make the practitioner cautious in the expression of his beliefs.

Treatment.—The treatment consists in the application of local agents and in appropriate systemic remedies. Guided by the mirror, solutions of nitrate of silver, sulphate of zinc, sulphate of morphine, carbolic acid, carbolate of iodine, and chlorate of potassium, can be applied to the affected area directly. Powders may be thrown in by insufflation—as alum, tannin, iodoform, bismuth, etc. Atomized fluids, consisting of solutions of the mineral and vegetable astringents above mentioned, may also be used. When there is much irritation of the larynx, penciling the interior with solutions of bromide of potassium, sulphate of morphine, and of cocaine are highly useful.

The usual means to improve the nutrition of the body are necessary. Cod-liver oil, the phosphates, and hypophosphites must be freely administered. Indeed, the general plan for the treatment of phthisis should be followed, as elsewhere laid down.

SYPHILIS OF THE LARYNX.

Pathogeny.—Syphilitic lesions of the larynx occur with other mucous affections in from two to six months after infection. Again, the larynx may be affected years after all manifestations have apparently ceased, in this respect behaving as syphilis does in any organ which may be attacked. The disease is invited to this organ by its functional uses, and hence occurs in those who employ freely the organ in speaking and singing.

All the various syphilitic mucous lesions occur in the larynx—as condylomata, gumma, etc. They ulcerate, destroy the mucous membrane, and light up perichondritis with ulceration of the cartilages.

Symptoms.—There is usually but little pain, although some soreness may be developed by pressure. Hoarseness is an early symptom, produced by catarrh or by ulceration. Complete loss of tone may result from destruction of the cords. More or less cough, often husky and stridulous in character, is present in most cases. Difficulty in swallowing occurs when there is much ulceration of the epiglottis and of the aryteno-epiglottidean folds.

The actual condition of the parts is ascertained by laryngoscopic examination. The condylomata appear on the vocal cords, on the aryteno-epiglottidean folds, and on the posterior wall of the larynx, as a flat, wart-like papule, covered with a grayish pellicle. The mucous membrane around it is hyperæmic and catarrhal. Gummata are deposits in the membrane, in size from a pin-head to a pea, and situated on the true and false vocal cords, on the epiglottis, and elsewhere. These syphilitic new formations soon ulcerate. The depth and extent of the ulcerations depend on the stage of the disease, for the syphilitic deposits lie the deeper the more chronic the duration of the constitutional state. The superficial lesions will heal without deformity, but, when gummata ulcerate, the destruction is so great that the most serious changes ensue in the form and structure of the organ.

Syphilitic ulcerations extend from the pharynx into the larynx by contiguity of tissue. Infiltration of the margins of the epiglottis are followed by ulceration and necrosis; then the aryteno-epiglottic folds are invaded, and ultimately the vocal cords are reached and destroyed. Extensive deformity may result in such cases from the contraction of the cicatrices, and the functions remain impaired permanently.

Course, Duration, and Termination.—The progress of the ulceration in these cases is much affected by the constitutional state, and by the treatment. If permitted to pursue its own course in a constitution depraved by excesses, and by repeated mercurialization, the destruction will be very great. Even extensive ulcerations are, however, remarkably restored by suitable medication. As respects the course, duration, and termination, everything depends on timely and appropriate treatment. When syphilitic perichondritis of the laryngeal cartilages takes place, the duration of the disease is increased, and its importance enhanced in every respect. A fatal termination may occur in these cases by œdema of the glottis or by hæmorrhage. The question of the relief to be expected by treatment is much influenced by the condition of the subject and by the amount of mercury previously taken.

Diagnosis.—There is no well-marked distinction between tubercular and syphilitic ulcerations, as respects the appearances seen on laryngoscopic inspection. The previous history and attendant circumstances possess a high degree of diagnostic importance. In the case of tubercular ulcerations the signs of phthisis accompany the laryngeal symptoms; in syphilitic diseases, mucous patches, cutaneous affections, nodes, etc., are present, or have appeared at some time since a chancre formed.

Treatment.—Syphiloma of the larynx proceeding to ulceration requires the most energetic handling to prevent irremediable damage. If a proper mercurial course has not previously been administered, it should be undertaken at once. The internal use should be conjoined with inunction of mercurial ointment and the local application of mercurial fumigation. If the deposits are gummata, no treatment is so efficacious as the rapid administration of iodide of potassium—from twenty to sixty grains being given every four hours, according to the urgency of the symptoms. When destruction of the vocal cords is threatened, no time should be lost by the exhibition of small doses. For the cases of secondary or tertiary disease, with superficial ulcers, sluggish in character, medium doses of corrosive chloride of mercury may be given with advantage. The vapor of iodine can be inhaled with good effects, and the tincture painted over the larynx is serviceable. Various combinations of iodine and mercury are prescribed in chronic syphilis, but, generally speaking, it is better to give these agents separately. In broken constitutions stillingia has good effects. Cod-liver oil is always useful when the nutrition is poor.

PERICHONDRITIS OF THE LARYNX.

Definition.—The cartilages of the larynx are occasionally affected by contiguity of tissue, the disease extending from the overlying mucous membrane. *Perichondritis* means an inflammation of the investing tunic, the perichondrium. There occur several forms—for example: *Perichondritis arytænoidea; P. cricoidea;* and *P. thyroidea.*

Pathogeny and Symptoms.—Inflammation of the laryngeal cartilages may be primary or secondary. Injuries and excessive exertion of the voice seem to be causative. The disease usually arises by the extension of tubercular or syphilitic ulcers to the perichondrium. Inflammation of the perichondrium is followed by softening and disintegration, or necrosis of the cartilage, swelling and stenosis of the larynx, the formation of an abscess, swelling of the neighboring lymphatics, the discharge of pus internally or externally, or in both directions, the formation of fistulæ, the separation of the necrosed cartilage, etc. There are special symptoms, determined by the particular cartilage affected, and general symptoms common to them all. The latter consist in cough, alteration of voice, or a toneless voice, enlarging lymphatics, and swelling of the larynx externally, but especially the symptoms of laryngeal stenosis, difficult breathing, suffocative attacks in paroxysms, a suppressed or stridulous cough, aphonia, etc. By a laryngoscopic examination, the seat of swelling may be ascertained, and the position and behavior of the vocal cords will indicate implication of the arytenoid and also of the cricoid. Perichondritis thyroidea may be internal or external. A case of the latter has happened under my observation in which a fistulous opening existed on the front of the trachea. A spot of necrosis probably had formed on the anterior plate of the cartilage. A fistulous communication may exist between the interior and exterior of the larynx, caused by an abscess breaking in both directions.

Treatment.—The prompt opening of abscesses and laryngotomy are very necessary measures when suffocation is imminent. In the very rare idiopathic form, recovery may ensue, even after the necrosis and removal of a cartilage, but, in the tuberculous cases, death is the usual result.

TUMORS OF THE LARYNX.

Forms.—Tumors of the larynx may be divided into the two great classes of benign and malignant. In the former are grouped *papilloma*, the most common form of tumor, having its seat in the larynx; *fibroma*, next in frequency, also known as fibrous polypi; *cysts*, or mucous polypi; *myxoma* and *lipoma*, which are very infrequent. In the malignant form is *carcinoma*, which is comparatively common and is usually primary—that is, begins in the larynx.

Causes.—Tumors occur in the larynx, in the great majority of cases, at the most vigorous period in life—from twenty to fifty. Carcinoma is a disease of advanced life, and develops from forty to sixty. Some tumors may appear early; for example, papilloma, which is found in children under twelve, but is more frequent at a later period. Men are much more liable than women, for in them disease of the larynx is invited by their avocations, by speaking and singing in the open air, by the irritation of cigarette-smoking, and by the inhalation of irritating dust, gases, etc. Carcinoma arises under circumstances with which we are unacquainted, unless heredity may be traced.

Pathological Anatomy.—Papilloma, a tumor of villous structure, develops from the vocal cords, epiglottis, and aryteno-epiglottic folds, but its favorite seat is the anterior portion of the vocal cord. It consists of branched connective-tissue bodies, containing numerous and large capillaries, and are covered by epithelium (Wagner). "The extensive papillomata that occasionally occur in the larynx in children are perhaps always of syphilitic origin" (Billroth). As seen by the laryngeal mirror, they are branched, cauliflower-like vegetations, which, to a greater or less extent, occupy the upper and middle laryngeal cavity.

Fibroma is a roundish tumor, dirty-white or red in color, and usually grows from a vocal cord. It is connected to the point of origin by a pedicle of greater or less length, and may have a considerable range of movement. It consists of connective-tissue fibers variously interlaced, is somewhat nodulated, abundantly supplied with vessels, but contains a small amount of fluid (Billroth, Wagner). It grows very slowly, and ultimately attains to great size.

Mucous cysts belong to the class of retention cysts. A duct of a follicle obstructed, the contents accumulate, and thus a tumor is formed, attached usually by a broad base, but ultimately becoming pedunculated. They grow in the ventricle of Morgagni, and at length project into the upper laryngeal cavity (Virchow).*

Of the carcinoma attacking the larynx, the epithelial greatly preponderates over the other varieties. According to Von Ziemssen, of sixty-eight cases of carcinoma, fifty-seven were of the epithelial

* "Die krankhaften Geschwülste," Band i, p. 246.

variety, nine encephaloid and scirrhus, and two villous. The disease first appears on the vocal cords and then extends upward to the aryteno-epiglottic folds. It occurs about equally on the two sides, and involves both ultimately.

Symptoms, Course, and Termination.—In the case of papilloma and fibroma of the larynx, the main symptom is that of a gradually increasing obstruction of the organ—an inspiratory dyspnœa, while expiration is easy. When the obstruction is considerable, inspiration is whistling, noisy, crowing, wheezing, there is constant feeling of a need of air, and the lips are rather bluish, the eyes prominent and injected. The earliest symptom is an alteration of the voice. Before the patient experiences any difficulty of inspiration, the voice has become raucous, hoarse, and finally toneless. There is also some cough, and this becomes husky and stridulous, and violent attacks of inspiratory dyspnœa occur with every paroxysm. A sensation of the presence of a foreign body, stuck fast in the throat, comes on as the neoplasm develops. In the further progress of the cases the dyspnœa increases, swallowing becomes more and more difficult, the nutrition suffers, and the strength declines. Unless relieved by operative procedures or, in the case of syphilitic papillomata, by iodide of potassium, the distress increases with the growth of the tumor, and a slow death is caused, accompanied by the horrors of a gradual suffocation.

Carcinoma of the larynx differs from this disease in other situations, by reason of the existence of a distinct prodromal symptom. Hoarseness precedes the development of the other symptoms by several years, and there appears to be a prodromal hoarseness lasting a year or more. Pain is also an early symptom, but this indicates the formal development of the local morbid process. The pain is situated deeply in the larynx, and is sometimes felt very acutely in the ear. A gradual decline in strength and weight, an earthy hue of the complexion, pearly sclerotics, a weak pulse, and breathlessness on slight exertion, are significant symptoms indicating the development of the cancerous cachexia. The cervical lymphatics begin to enlarge about six months after there are symptoms distinctive of laryngeal disease. The larynx usually enlarges somewhat and is tender to pressure. When perichondritis occurs, extensive suppuration and œdema greatly add to the dimensions of the larynx, as they increase the difficulty in breathing. Some time after the hoarseness, but not long after pain has begun, the symptoms of laryngeal stenosis occur. At first the dyspnœa is excited only by exercise, but after a time becomes constant and most distressing. Cough comes on with the first change in the larynx, and after a time bloody mucus and, in some cases, considerable blood are brought up. Violent paroxysms of dyspnœa occur with the cough, with the inspiratory efforts especially. The cough becomes husky and stridulous with the progress of the changes in the vocal cords. Pain in swallowing occurs soon after the disease manifests itself in the larynx, especially when the epiglottis and the aryteno-epiglottic folds are attacked. Considerable destruction of the parts about the entrance of the larynx entails great suffering in attempts to swallow, and, the entrance to the glottis not being properly protected, particles of food and drink drop into the larynx, exciting violent paroxysms of coughing and dyspnœa.

The usual duration of carcinoma of the larynx is about two years. Cases have terminated earlier, and others have lasted from five to ten years, but these are exceptional. Removal of the larynx has prolonged life, but the ultimate termination is in death.

Diagnosis.—The differentiation between the several forms of benign tumors is arrived at by the laryngoscope, so far as the naked-eye appearances will solve the question. To distinguish between carcinoma and the benign growths, attention must be given to the following points: the age of the subject, the appearance of a tumor after a long period of hoarseness, the occurrence of rather severe pain in the larynx and in the ear, the enlargement of the cervical lymphatics, and the gradual development of the cancerous cachexia—such are symptoms of carcinoma of the larynx, and to these must be added the ordinary signs of a tumor. To differentiate between carcinoma and syphiloma, the history of the case becomes essential, for there are no means of separating ulcerating gummata from epithelioma on inspection by the unassisted eye. The administration of some large doses of iodide of potassium will, by the results which follow, illuminate the character of the case. This remedy, or its therapeutical congener, mercury, will be necessary to separate syphilitic papillomata from the simple form.

Treatment.—As Billroth finds that papilloma occurring in children is apt to be syphilitic, and as the differentiation of certain ulcerations of the larynx also require it, large doses of iodide of potassium, or suitable mercurial remedies, should be administered at the outset. This failing, removal of the neoplasm becomes necessary. There are two methods—endo-laryngeal extirpation, and removal by laryngo-tracheotomy. After a course of manipulation, which has for its object the removal of the sensitiveness of the larynx and fauces, the growth is removed by the application of caustics (chromic acid), by the cutting-forceps, guillotine, wire *écraseur*, etc., or by the galvanic loup, or cautery.

SPASM OF THE GLOTTIS—PSEUDO-CROUP—LARYNGISMUS STRIDULUS.

Definition.—*Spasm of the glottis* is a term applied to spasm of the muscles of the larynx, innervated by the recurrent or inferior laryngeal nerves. The mechanism consists in an irritation of the terminal filaments of the pneumogastric in the mucous membrane of the larynx, the transmission of this irritation to the pneumogastric nucleus, and its reflection over the motor nerves supplying the laryngeal muscles.

Symptoms and Pathogeny.—Spasm of the glottis is never the initial symptom. For the first day or two, the child suffers from a simple acute catarrh. There may be slight feverishness, but not high fever; there is more or less nasal catarrh ; the eyes are apt to be injected; the throat is redder than normal ; the voice is a little hoarse, and there is some cough—in fact, the symptoms are those of an acute cold. Toward evening the voice may get hoarser, and the cough assume a more ringing tone. But in the night the child awakes rather suddenly, coughing in the brassy, metallic, resonant tone which is called "croupy." Every strong inspiration is accompanied by a loud, crowing stridor, and on crying each inspiration has the same character, the expirations being wheezy and somewhat stridulous. This peculiarity of the inspiration is due to sudden and high tension of the vocal cords, they being approximated, and consequently narrowing the chink through which the air passes. So difficult is the entrance of air, that the accessory muscles of respiration are brought into use, the alæ of the nose work convulsively, the face and lips are somewhat bluish, the countenance is anxious, and the inferior portion of the chest is drawn in instead of being expanded during inspiration. Such is an ordinary case of pseudo-croup. Undoubtedly, there are examples of the disease in which the point of irritation is the stomach. An indigestible supper, or some improper article eaten during the evening, may set up an irritation of the end-organs of the pneumogastric, which may be reflected over the laryngeal motor nerves, producing the symptoms of laryngismus stridulus. In which mode soever produced, spasm of the glottis quickly subsides under appropriate treatment, and in an hour or two after being awakened by the oppression the child is usually sufficiently relieved to become drowsy, barking in its sleep, occasionally, until the morning. This experience may be repeated on the following night, and indeed for several nights. When this recurrence of the paroxysms takes place, the case awakens renewed anxiety, lest an exudation may be forming in the larynx. If the paroxysms recur for two nights, there will be attacks during the day also. The author has observed a few cases in which the spasms continued for several days ; without being violent at any time, the cough had always the "croupy" character, and a strong inspiration developed stridor.

Course, Duration, and Termination.—The simplest cases consist of a mild acute catarrh, inducing a nocturnal attack of spasm of the glottis, which terminates in an hour or two. The catarrh soon subsides, and there is no return of the spasm of the glottis until succeeding attacks of catarrh renew the disturbance in the nervous apparatus of the larynx. As only certain children, though by no means a small proportion, suffer, there is probably a peculiar mobility of the nervous system necessary.

As the mobility of the nervous system is much more pronounced in children than in adults, we have in this an explanation of the fact that spasm of the glottis is a disease of early life, and rarely occurs after twelve. Although a malady of little importance, spasm of the glottis accompanies some of the most serious diseases. Thus it occurs during the course of true croup, diphtheria, œdema of the glottis, etc., and may be the immediate cause of death; and in all cases adds materially to the difficulties, by the frequent spasms in the laryngeal muscles. As it usually occurs in children, arising in a reflex disturbance, having its origin in an acute catarrh, or an acute indigestion, it always ends in recovery. There are occasional (rather rare) cases in which the catarrh terminates in œdema of the glottis.

Diagnosis.—The manner of its occurrence and the promptness of the cure sufficiently indicate the nature of pseudo-croup without the laryngeal mirror.

Treatment.—Formerly, every case of the disease was subjected to a severe ordeal, and, when bloodletting and tartar emetic were abandoned, emesis was still persevered in. No perturbating agents of this kind are really necessary. A few drops of the fluid extract of ipecac, given every twenty minutes until nausea is produced, will relieve if a cold wet pack about the neck has failed. From five to twenty grains of the bromide of potassium will usually succeed, and will be more effective if some chloral is added. From ten minims to ʒj of paregoric often arrests the paroxysms. A minute dose of pilocarpine nitrate or muriate ($\frac{1}{40}$ to $\frac{1}{8}$ grain) will stop the spasms usually when diaphoresis begins. As it is so mild a disease, the simplest means will suffice to cure an attack. Children accustomed to the attacks should receive prophylactic treatment. A daily morning cold bath to diminish the susceptibility to colds, the sirup of the iodide of iron, or the lactophosphate of lime, to promote the body nutrition, suitable clothing, and outdoor occupation, are the most approved means to prevent a recurrence of the seizures.

CROUPOUS LARYNGITIS—TRUE CROUP.

Definition.—The preponderance of authority is in favor of that view that the so-called membranous croup is only laryngeal diphtheria. The author is one of those who maintain that *croupous laryngitis*, or membranous croup, is an independent, substantive disease; that we have a croupous laryngitis as we have a croupous bronchitis and a croupous enteritis. The author believes that this disease is distinct and separate from diphtheria, for the following reasons: it occupies the larynx exclusively, is a purely local affection, the exudation is *on* and not *in* the mucous membrane, and that systemic poisoning, or secondary septicæmic and infective embolic processes never result from it.

Causes.—Croup is a disease of childhood, and very rarely occurs later than the second dentition, and attacks male children by preference, in the proportion of three to two. It is not merely the ill-fed children of the poor, or the inheritors of scrofula and rickets, who are chiefly attacked, but the vigorous and well-nourished are more liable. It is certain that heredity has an important influence in its causation, in that certain families are especially liable to destructive visitations, and others, living under similar conditions, escape. Notwithstanding the prevalent opinion that humidity, coldness, and variability of climate favor the development and spread of croup, we find that Lombard says "he has sought in vain to discover any difference in the development of this disease as regards climate, latitude, and altitude."* It seems, nevertheless, well established, that humidity favors its occurrence, and that more cases occur in winter and spring than in summer. That true croup prevails as an epidemic is highly improbable, but, as diphtheria does, the error, if it exist, has arisen by confounding the diseases. A croupous laryngitis sometimes arises during the course of the acute infectious diseases, as measles, scarlatina, small-pox, etc., but of measles especially. This may be a diphtheritic process superadded to an existing lesion, but is more probably a mere croupous inflammation.

Pathological Anatomy.—The initial hyperæmia is of an intense character; the mucous membrane is swollen, has a deep-red color, is marked by an exceedingly fine but diffused arborescent injection, and here and there by minute ecchymoses, and the sub-mucous connective tissue is more or less œdematous. In the progress of the case the redness subsides to a large extent, but the membrane continues somewhat thickened for some time longer. Soon after the hyperæmia attains its maximum, there appears on the surface of the inflamed mucous membrane a grayish, semi-transparent pellicle, which soon becomes thicker, grayish-white, yellowish, or brownish—an opaque false membrane. At various places the false membrane differs in coherence, density, and adhesiveness: here, several lines in thickness, uniform in structure, and firmly attached to the mucosa; there, in flakes or patches, loosely attached to the surface beneath. The false membrane is found on the vocal cords throughout their whole extent usually, spread over the ventricles, and attached to the inner surface of the epiglottis. There may be none found *post mortem*, it is alleged; but probably in these examples there was an error of diagnosis. Successive deposits—two or three—may occur; the first exuded is softened by the serum which transudes, as does the albumen, and is mechanically detached in the act of coughing. As expectorated it usually appears in the form of grayish-white shreds or casts, several lines in thickness, and tolerably tough. Sometimes a cast of the trachea and tubes of considerable extent is thrown off, but this is exceptional. On microscopic examination, the false membrane is found to be composed of a fine network of fibrillæ, holding in their interstices leucocytes, and chemically of an albuminous nature, or of fibrin. Soon after the false membrane forms on the epithelial surface of the mucosa, a process of detachment begins, by the accumulation of serum, having suspended in it muco-pus, cast-off epithelial cells, blood-corpuscles, etc. The mucous membrane, when the exudation is detached, is found to be unaffected, except the hyperæmia, and the imbibition of fluid affecting the epithelial cells. In this absence of direct implication of the epithelium lies the distinction between croup and diphtheria, for in the latter the false membrane is closely united to, and is probably developed from, the cells of the epithelium, as E. Wagner has apparently shown. After the exfoliation of the first croupous exudation, there may be several successive crops of exudation, or, ceasing to form again, a cure is effected. The false membrane is not confined to the parts on which it first appears, but extends upward into the pharynx, but especially downward into the trachea, primary bronchi, and smaller bronchi. As the membrane extends toward the finer tubes, it becomes less fibrillary and more cellular, until at length it is a mere muco-purulent fluid. The lungs are affected by emphysema, and here and there atelectasis, the result of the inspiratory obstruction and the tenacity of the exudation blocking some of the finer tubes.

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* "Traité de Climatologie Médicale," etc., tome iv, Paris, 1880, p. 401.

Symptoms.—The attack of croup usually but not invariably begins as an acute catarrh of the larynx ; there is a feeling of heat and irritation in the organ, and the voice is a little husky ; there is cough with something of stridor about it, and fever, restlessness, thirst, anorexia, and disturbed sleep, accompany the evidences of laryngeal mischief. When the fauces are inspected, more or less redness, sometimes dusky redness, will be observed, and also small patches of a thin, pellicular exudation of a grayish-yellow color, studded over the palate, tonsils, and pharynx. These patches presently coalesce and then form a denser membrane several lines in thickness, of a yellowish-gray or ash color. As huskiness of voice was one of the initial symptoms, the same patches of pellicular exudation are forming in the larynx. Although it is affirmed of croup that the exudation spreads sometimes over the tongue, cheeks, lips, into the nose, ears, etc., these cases so behaving are examples of diphtheria, it is most probable, for true croup does not extend beyond the pharynx and soft palate. The submaxillary glands become somewhat tumid and swollen, but not the chain of cervical glands extending under the sterno-cleido-mastoid muscles, which are enlarged in diphtheria. Usually from one to two days are occupied with the development of the catarrhal form, but other and rare cases commence with abruptness in the night, as an ordinary spasm of the glottis. In what mode soever developed, there now later than the second dentition, and attacks male children by preference, in the proportion of three to two. It is not merely the ill-fed children of the poor, or the inheritors of scrofula and rickets, who are chiefly attacked, but the vigorous and well-nourished are more liable. It is certain that heredity has an important influence in its causation, in that certain families are especially liable to destructive visitations, and others, living under similar conditions, escape. Notwithstanding the prevalent opinion that humidity, coldness, and variability of climate favor the development and spread of croup, we find that Lombard says "he has sought in vain to discover any difference in the development of this disease as regards climate, latitude, and altitude."* It seems, nevertheless, well established, that humidity favors its occurrence, and that more cases occur in winter and spring than in summer. That true croup prevails as an epidemic is highly improbable, but, as diphtheria does, the error, if it exist, has arisen by confounding the diseases. A croupous laryngitis sometimes arises during the course of the acute infectious diseases, as measles, scarlatina, small-pox, etc., but of measles especially. This may be a diphtheritic process superadded to an existing lesion, but is more probably a mere croupous inflammation.

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Now and then, on sudden, deep inspiration, there is still the peculiar whoop, but the voice becomes more and more husky. Dyspnœa now comes on. The respirations increase in frequency, and are seen to be so labored as to require the aid of all the muscles. The child can not lie down. If, exhausted by the efforts made, the child seeks repose, resting its head high upon a pillow, it soon starts up in a fright, breathing more heavily, and with a shrill, whistling inspiration. Tossing from side to side, he seeks, in endless changes of position, for the relief which no change brings. With open mouth, rapidly working alæ of the nose, and every respiratory muscle called into play, he exerts himself to the utmost to obtain the necessary air, but ineffectually, the lower portion of the chest being drawn in deeply with each inspiration. The air passes with difficulty through the narrowed chink of the glottis, and hence the slowness, and the whistling, crowing, and stridulous inspirations, which can be heard at quite a distance from the patient. Ultimately the narrowing of the glottis is such that expiration becomes difficult and somewhat noisy. To the difficulty of breathing from the swelling of the mucosa and the presence of the false membrane are now added paroxysmal attacks of spasm of the glottis. When these attacks come on, suffocation seems imminent. The child, who has been restless when these seizures are felt, tosses wildly about with an agonized expression, tears at his throat to remove some obstacle, the face cyanosed, the alæ of the nose widely separated, the inspiratory efforts gasping, and the muscles working to their utmost, the body covered with a profuse sweat from the intensity of the exertions; and at last, when death seems at hand, a little air enters the chest, the breathing becomes somewhat easier, and the child, exhausted and stupefied by the carbonic acid which is accumulating, drops into a fitful sleep of a few minutes' duration. These suffocative attacks appear at shorter intervals. By some these attacks are supposed to be due to a paresis of the laryngeal muscles instead of spasm, and Steiner supports the opinions of Niemeyer on this point. In some cases there occur decided remissions between the attacks of suffocative dyspnœa. Considerable portions of false membrane being expelled, air again enters the lungs; the cyanosis disappears, the fever ceases, and some refreshing sleep is obtained. As the false membrane is renewed again, the former difficulties are resumed; the breathing becomes difficult, and the suffocative attacks even more violent. Sometimes a mass of exudation is suddenly detached and thrown against the under surface of the vocal cords; breathing is suspended, the child turns deeply blue in the face, and violent coughing sets in, detaching the mass, and either carrying it down by inspiration, or outward by an explosive cough. In the cases which tend to a favorable termination, the appearances of improvement, noted between the suffocative attacks, are maintained. The paroxysms of suffocation become less frequent, and the constant dyspnœa visibly lessens; the cough has less and less of the barking character, and the expectoration is more abundant and looser; the fever disappears; the voice gradually passes from toneless to husky and loud; sneezing occurs, and the nose discharges. If, instead of improvement, the case goes on as usual to a fatal termination, the final stage of *asphyxia*, or carbonic-acid poisoning, is now entered on. The cyanosis deepens, the agonized expression of countenance is replaced by indifference, drowsiness, and stupor, the eye grows dull and is nearly closed, the difficulty of breathing continues, and the respirations are frequent and shallow, but without the whistling and stridor. Now and then a paroxysm of dyspnœa comes on, in which the child is roused from its somnolent condition, gasps for breath, struggles, and then lies down, passing at once into an apathetic state. The symptoms of vital failure now come on: the pulse becomes rapid and weak; a cold, clammy sweat covers the body; the extremities are cold, the somnolence deepens into stupor and insensibility, carpopedal contractions occur, and sometimes general convulsions.

Course, Duration, and Termination.—The first stage, characterized by the symptoms of laryngeal catarrh, runs its course in twenty-four to thirty-six hours. The fulminant cases, beginning abruptly at the second stage, with its symptoms of laryngeal stenosis, will terminate fatally within two days, and sometimes within one day. The usual duration of ordinary cases is about one week, and rarely do cases extend to ten days. The second stage may continue from one to fourteen days, but the latter duration must be regarded as exceptional. The third—the stage of asphyxia—lasts from thirty-six to forty-eight hours. In most of the cases the cause of death is general paralysis, due to carbonic-acid poisoning. Very rarely is death caused by apnœa, the access of air prevented by closure of the glottis with shreds of false membrane, or by spasm. Œdema of the glottis, croupous pneumonia, œdema of the lungs, or capillary bronchitis, may be a cause of death.

Diagnosis.—Until the characteristic membranous formation appears in the throat, croupous laryngitis may be confounded with pseudo-croup or laryngismus stridulus. The latter occurs frequently in some children, comes on suddenly in the night, and after a few hours ceases to give trouble. True croup develops more slowly and does not present the apparent laryngeal obstruction of false croup until the case is well advanced. The fulminant form, it is true, begins abruptly and with violence, but there is no amelioration in the condition as in pseudo-croup. The most certain means of diagnosis consists in the discovery of the exudation, which soon appears after the initial symptoms are well declared.

Treatment.—The means employed in the treatment of membranous laryngitis are naturally divisible into two classes—local, systemic. An almost infinite variety of remedies have been applied to the throat: we mention those that are really useful. Caustic applications, as nitrate of silver, the mineral acids, etc., are injurious; for, although they may remove the existing membrane, they can not prevent its reformation, and the extension of the exudation is invited to the healthy tissue corroded by the caustic. Solvents that are not irritating are most useful. The first and most important one is lime-water, which may be applied by a large soft probang, or atomized by a spray douche. The application of the spray should be nearly continuous; of the probang, frequent. An excellent method consists in slaking bits of freshly burned lime in water placed in a wide-mouthed bottle—the patient inhaling the vapor as it arises. Next to lime-water is lactic acid, as a solvent, and it is as safe as it is efficient. Sufficient of the acid should be added to water until a distinctly sour solution is obtained, and this may be freely applied by the spray douche or probang. Recent reports are very favorable to washed sulphur or sublimed sulphur freely dusted over the affected parts in diphtheria. Chlorate of potassa is preferred by many, either atomized or on probang or brush; it is also used with chloride-of-iron tincture, or the latter, undiluted, is applied on a camel's-hair brush to the false membrane and fauces. The bromides of potassium and ammonium, in solution, are also sprayed over the throat and fauces. Good results have been claimed for a mixture of fluid extract of belladonna and the bromides in solution, used in the same way, a continuous application of the spray for hours at a time, or until the pupils are affected. It is claimed for this mixture that the belladonna allays the spasms of the glottis. A solution of chloral has been employed as a local application, both for its antiseptic effects and as a moderator of the reflex spasms of the laryngeal muscles. The air of the apartment should be kept moist with the vapor of water, or impregnated with the vapors of eucalyptus and turpentine oils, which can be accomplished by boiling in a suitable vessel some eucalyptus leaves with turpentine. This expedient has been found to be exceedingly useful in the Children's Hospital at Lisle.* The internal remedies are equally numerous. There are three main objects to be kept in view in the treatment of true croup: to detach, remove, and prevent the formation of the false membrane; to prevent the attacks of laryngeal spasms; to maintain the strength. Quinine, calomel, chlorate of potassa, tincture of iron, and the bromides, are recommended, and some of them much lauded by their respective proposers. There are two of unquestionable utility—quinine and bromide of ammonium. Quinine should be administered in full doses (for a child, three to five grains every three or four hours). Cinchonism should be kept up as fully as possible, with the object to stop the fibrinous exudation. In alternation with quinine, or by itself, should be administered full doses of bromide of ammonium. The particular fact which gives value to this and the other bromides is its elimination by the bronchial and faucial mucous membrane, thus acting locally. Furthermore, quinine and the bromides check the spasm of the laryngeal muscles, a most important action. The mechanical effect of an active emetic is often necessary to dislodge the obstructing membrane. Apomorphine is especially effective for this purpose. Ipecac is too depressing, tartar emetic is highly objectionable; alum and subsulphate of mercury are the best. According to Barker, of New York, the subsulphate has special power as a remedy for croup, an opinion in which the author is disposed to share. It should be given early, and not wait for severe obstruction. Besides the agents above advised—quinine and the bromides—for the laryngeal spasms chloral is to be commended. The author has preferred to give chloral and bromide of ammonium together, and the quinine separately. Besides its power to allay the spasms, chloral is one of the few remedies which possess the property to check the formation of an exudation. Many practitioners hold that chlorate of potassa has this property (Steiner), and this remedy is probably more largely prescribed than any other in croup and diphtheria. There are practitioners who still hold to the aplastic virtues of calomel, and use this remedy in large doses, with asserted success, but the most approved authorities are opposed to both opinion and practice (Oppolzer, Steiner). The measures to maintain the strength are very important. Alcoholic stimulants possess, according to the Brooklyn physicians, some peculiar, possibly specific curative power. It is alleged that the best results are obtained in diphtheria by large and sustained administration of whisky, brandy, etc. How far these facts are applicable to true croup remains to be determined.

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* "Journal de Therapeutique," February, 1886.

CORYZA—NASAL CATARRH.

Definition.—By the term *coryza* is meant a catarrhal inflammation of the nasal mucous membrane. It may be either *acute* or *chronic.*

Causes.—Atmospherical causes are the most frequent and influential. The exposure of the neck to a current of cold air, of the feet and ankles to cold and dampness, passing from a warm to a cold atmosphere, and from a cold to a warm atmosphere suddenly, are among the most usual causes. Irritating gases and vapors, the spores of some plants, certain powders, as ipecac, tobacco, etc., excite an irritation of the nasal mucous membrane. Heredity is an occasional factor. Epidemic influence now and then prevails on an extensive scale.

Pathological Anatomy.—An intense hyperæmia is the first change, with an arrest of secretion. This is soon followed by swelling or tumefaction of the membrane ; the epithelium is detached, and a great number of new cells are produced. The mucous glands furnish an abundant secretion very rich in saline constituents. If the congestion is intense, vessels are ruptured, and more or less epistaxis results. With the progress of the case, a change occurs in the character of the discharge ; at first watery and transparent, it becomes thicker and opaque with the increase of the pus-cells (leucocytes). When recovery takes place, the secretion diminishes, the congestion subsides, and the swelling of the membrane disappears. Such is the usual course of an acute inflammation. In the chronic form, the mucous membrane is reddish-brown, in very old cases grayish, the veins are dilated and varicose, often forming polypoid protrusions. There may be more or less extensive ulceration, and losses of substance, in old cases. The discharge is thick, greenish, and often offensive from decomposition. Large collections of inspissated mucus form on the turbinated bones.

Symptoms.—Taking cold in the head is announced by chilliness, weariness, headache, and general muscular soreness. The nares are dry, feel stuffed and uncomfortable, and an inclination to sneeze is often felt. Presently the nose pours out an abundant watery and saline discharge, the anterior nares are red and inflamed, and sneezing is frequent. The discharge soon assumes a purulent character, and contains numerous micrococci. The voice has a peculiar tone, rather nasal and muffled from the swelling of the nasal mucous membrane. In a few days the swelling subsides, the secretion lessens, and health is restored in about two weeks from the beginning of the attack. The chronic form may grow immediately out of the acute affection, or it may be the result of repeated acute attacks, or develop from the continued operation of the causes. In the chronic form of the disease, the mucous membrane is either livid, the vessels varicose, and the connective-tissue basis of the mucous membrane hypertrophied, or the membrane is pale, thin, bloodless, and atrophied. The discharge consists of greenish, offensive pus, or of scales taking the form of casts of the bones, which are also offensive from decomposition. If the mucous membrane is destroyed by ulcerations, and caries of the bones has occurred, the case is then called *ozæna*. The morbid process extends through the nasal passages and into neighboring cavities.

Course, Duration, and Termination.—The acute form reaches its maximum in a few days, and terminates in from fourteen to sixteen days if uninterfered with. The chronic form is excessively obstinate, and continues with varying fortunes for several years. During the summer and autumn it is milder, but in the winter and spring it is worse. Although there is no danger to life, the disease in its chronic form is difficult to cure. The popular notion that extension to the lungs takes place is entirely unfounded. In the phthisical, the coexistence of nasal catarrh and the pulmonary lesions, which is very common, is often supposed to mean the dependence of the latter on the former.

Treatment.—An existing constitutional dyscrasia, especially syphilis, needs attention. If the least suspicion may be entertained, an iodide-of-potassium course should be carried out. Where there is a strumous diathesis, cod-liver oil, the phosphates, iodide of iron, etc., should be employed. If we have to deal with an attack of acute catarrh, an attempt may be made, and will often prove successful, to abort it by the administration of a full dose of quinine and morphine (for an adult, gr. xv of quinine and gr. ss. of morphine). When established, the best remedy is Lugol's solution, one drop every hour or two. If there is fever, one drop of tincture of aconite-root every hour will prove efficient. If the secretion is watery and profuse, tincture of belladonna may be given with the aconite, two drops every two hours. In the local treatment of chronic catarrh, the first step necessary is to clear the mucous surface of adherent discharges. The nasal douche, so much employed, has so often given rise to inflammation of the middle ear, by forcing the application into the Eustachian tube, that it must be used with caution. The post-nasal syringe and tepid water containing a little common salt are the best materials for cleansing the passage. Numerous are the kinds and forms of applications—gaseous, liquid, and solid. The volatile applications consist chiefly of iodine and carbolic acid, separately or in combination. The tincture of iodine and carbolic acid may readily be volatilized and inhaled from a small bottle. Still more generally successful is the introduction of a cocaine tablet. Containing ⅛th or ⅙th grain in the form of a flattened disk, the tablet is introduced alongside the septum, where it is allowed to dissolve, the head being inclined backward to permit the medicament to apply itself to the affected surface. One should be introduced on each side of the nares, twice or three times a day. The effect is usually prompt and complete. These tablets are the more effective the earlier they are applied. The liquid applications consist of solutions of chlorate of potassa, chloride of ammonium, sulphates of zinc, cadmium, and copper, acetate of lead, etc. The solutions must be very dilute, not stronger than one grain of sulphate of zinc to four ounces of water, for example, because of the very sensitive condition of these parts. When there are great thickening and ulceration, requiring strong applications, they must be made with the guidance of the mirror, and be confined to the part diseased. The most effective application, according to the author's experience, is a powder composed of tannin and iodoform (ʒ j—gr. x) applied by means of an insufflator. The membrane must be first cleansed, then the powder is dusted over the diseased part, using a very small quantity. Pressure by means of a graduated series of bougies is a valuable mode of treating those cases in which the membrane is much thickened.

EPISTAXIS—NASAL HÆMORRHAGE.

Causes.—The Schneiderian mucous membrane is abundantly supplied with blood-vessels and bleeds easily. *Epistaxis* may be caused by ulceration of the membrane, by vascular tumors, by traumatism, by a constitutional state—the hæmorrhagic diathesis—by irritation of the mucous membrane, and by mechanical causes, as valvular disease of the heart, and the pressure of an intra-cranial growth, etc.

Symptoms.—There may be a sense of fullness of the head, headache, noises in the ears, vertigo, precede the epistaxis, and be relieved by it, or the bleeding may occur without any previous symptom to indicate its approach. The blood may at first be observed on the handkerchief; a sense of moisture about the nares suggests the necessity of blowing the nose, and then blood is seen coming drop by drop, and from a single nostril. The blood may be discharged by the posterior nares and be expectorated. On inspection of the fauces, it will be seen trickling down the soft palate and uvula, which will prevent the mistake of supposing it comes from the lungs. The quantity of blood discharged varies greatly. In most cases an ounce or two is lost, when the flow spontaneously ceases; again, many ounces—a pint, a quart even—may be lost, completely blanching the patient, and only ceasing because of the faintness. If the bleeding occur in a subject of the hæmorrhagic diathesis, it may continue to faintness and be resumed again as soon as the circulation regains its force. Under these circumstances epistaxis may endanger life. Again, epistaxis may occur periodically, as a manifestation of malaria, or take the place, vicariously, of the menstrual or hæmorrhoidal flux. Those cases due to the pressure of a tumor on the cavernous sinus, or pterygoid plexus, are accompanied by swelling of the eyelids, injections of the eyes, retinal changes, and the symptoms proper to tumor of the brain.

Diagnosis.—There can be no difficulty, if the inspection is made when the blood is flowing, in determining the source of the hæmorrhage. When, however, the bleeding occurs in sleep, from the posterior nares, and is swallowed, there may be, if vomiting of the blood occurs, much difficulty in ascertaining the true source. But the absence of any evidence of stomach ulcer and the occasional occurrence of nose-bleed will suggest the means of differentiation. The same method of analysis will be equally applicable to the apparent expectoration of blood, for the absence of pulmonary disease and the occasional occurrence of epistaxis will decide the probability in favor of bleeding at the nose.

Treatment.—The application of cold, in the form of ice, small pellets of which may be introduced into the nares, while a block of ice hollowed out to fit the nose may be put on outside, will often be sufficient to arrest the bleeding. Pressure on the artery supplying the anterior nares may be easily effected by passing the little finger under the lip, near the middle line where the artery may be felt. Simply pressing the nares together, to enable the blood to coagulate, may often suffice. If pressure and cold fail, a solution of tannic acid, or of alum, or of acetate of lead, may be thrown into the nares, and, if these fail, a solution of Monsel's salts. The measures above advised may be supplemented by the hypodermatic injection of ergotin, if necessary, and by the stomachal administration of arterial sedatives, as veratrum viride and digitalis. All other expedients failing, the posterior nares must be plugged.

DISEASES OF THE KIDNEY.

THE URINE—ITS COMPOSITION AND PATHOGENIC RELATIONS.

Organic and Inorganic Constituents.—The urine is a complex fluid which represents the waste of certain organic and inorganic constituents of the body. Full knowledge of its composition is, therefore, necessary to comprehend the metabolism of the tissues. Also, as the urine contains the products of the pathological changes occurring in the kidney, it is obvious that, to know these processes aright, it is imperative to ascertain the variations from its normal composition and character.

A healthy, fully developed adult passes from forty to fifty ounces of urine in twenty-four hours. But the quantity must always be considered relatively to the quality, or the urinary water must stand in a certain ratio to the urinary solids. In round numbers, the quantity of solid matter contained in normal urine is about 4 per cent. A simple rule for approximately determining the amount of solids in any given sample consists in doubling the last two figures of the specific gravity. Thus, if the specific gravity be 1,020, by multiplying 20 by 2, we have 40—or 40 parts in 1,000, which is 4 per cent. The whole amount of urinary solids excreted in twenty-four hours is, hence, merely a question of proportion : for example, if 40 parts of solids be contained in 1,000 parts of urine, how much in the daily quantity of urine discharged in the case under consideration ?

The solids of the urine consist of organic and inorganic matter. The following are the constituents of the organic matter: *urea, uric acid, hippuric acid, kreatinin*, traces of *phenylic, oxalic, lactic*, and other acids of the *aromatic series*, and *coloring matters* (urobilin or urohæmatin, or urochrom; uroxanthin or indican).

The inorganic constituents consist of *phosphates* (alkaline and earthy), *sulphates*, and *chlorides*. Minute quantities of unoxidized sulphur and phosphorus are, also, present in normal urine.

In the examination of urine the following points are to be considered: the *quantity* passed in twenty-four hours relatively to the *solids* contained in it; the *color*, clearness or cloudiness; the *odor;* the *reaction* (acid, alkaline, or neutral); the *specific gravity;* the presence or absence of visible *solid matters* (uric acid, urates, mucus, blood, etc.).

The quantity of urine passed in twenty-four hours should be accurately measured, and a specimen of the whole should be reserved for examination.

In every case, whenever practicable to do so, the morning urine—that passed on rising—and the evening urine—that passed several hours after the principal meal of the day—should be submitted to examination.

The *quantity* in the normal individual is from forty to fifty fluid-ounces. The *color* is that of straw, of amber, or is pale yellow. Urine should be completely transparent, or at most cloudy only after cooling, in consequence of the precipitation of the urates. The *reaction* of urine is acid. The diet may affect this strictly within normal conditions, as for example under a vegetable diet, exclusively, the reaction becomes neutral or even alkaline. The *specific gravity* ranges from 1,004 to 1,030, according to the age of the subject, the character of the diet, and, especially, the temperature of the air, for when the cutaneous transpiration is abundant, the amount of urinary water is proportionally lessened.

The composition of the urine in disease varies in respect to the proportion of its normal constituents, and by the appearance of new substances. Both require some consideration.

The Organic Constituents—Urea.—Normal urine contains about three per cent. of urea, and the average daily excretion of a medium-sized adult is somewhat more than one ounce—about five hundred and forty grains. The amount present is increased in febrile affections. In diseases of the liver, with alteration of its structure and especially with considerable loss of the substance of the organ, the quantity of urea excreted is considerably less than normal. In cases of albuminuria, also, the percentage of urea is low, because the secreting structure of the kidney is damaged, but it is found in the fluid of dropsy, and in other fluids, in considerable quantity, although the amount produced is below the normal.

Several processes for the quantitative determination of urea have been proposed, but only those capable of ready adaptation to clinical purposes need be mentioned here. Heretofore the process of Liebig has been most generally employed. This method is based on the fact that urea is precipitated by nitrate of mercury from its solution—an insoluble compound of mercury and urea being formed. The phosphates, sulphates, and carbonates are first precipitated by means of a saturated solution of baryta ; then, on slowly adding the mercuric nitrate solution, a compound of this reagent and the urea is thrown down. With standardized solutions, the proportion of urea can be readily estimated.

A still more convenient process for ascertaining the quantity of urea, at least approximately, is that devised by Dr. Squibb, of Brooklyn. It is known as the "hypochlorite process," and consists in decomposing the urea by the official solution of chlorinated soda (U. S. Pharmacopœia) and collecting the escaping nitrogen, from the quantity of which can readily be determined the amount of urea present. Although not absolutely accurate, this method affords a sufficiently close approximation for all practical purposes. Another comparatively convenient method is called the "hypobromite," and is, as the Squibb process, based on the decomposition of the urea and collection of the nitrogen. A very simple apparatus for carrying out this method has been devised by Dr. W. H. Greene,* of Philadelphia, and a similar arrangement has since been proposed by Dr. Doremus, † of New York. The liquid to effect the decomposition of the urea, and separate the nitrogen, is a solution of bromine in caustic soda. For exact and detailed descriptions of these several processes, the reader is referred to the recent works treating of the composition and chemistry of the urine.

Uric Acid and Urates.—The whole amount of uric acid excreted in twenty-four hours does not exceed twelve grains, and may be as low as five grains, and between these extremes it oscillates in healthy adults. It is extremely insoluble in water, and is precipitated as the urine cools. The forms assumed by uric acid are so characteristic that it is readily recognized on microscopic examination. (See *post.*) In a normal condition uric acid is not found in the blood, but in gout it is either produced in excessive amount, or fails to be oxidized into urea and thus excreted. The source of uric acid remains obscure. The most widely entertained theory regards it as a substance intermediate between albumin and urea, and when present in quantity signifies deficient oxidation. When the urine is highly acid, and concentrated, uric acid is in excess.

The word *urates* signifies a cloudiness or sediment composed of combinations of uric acid with various bases. The most common are the urate of soda, urate of ammonia, and urate of calcium. The least abundant is urate of potassium. Even when abundant, the urates are usually held in solution at the temperature of the body, to be cast down when the urine cools, then forming an abundant milky or chalk-like deposit, which may indeed have a brick-dust appearance, especially should uric acid be in excess. On heating such urine, the deposit or sediment clears up—a reaction which serves to distinguish between the urates and albumin, or phosphates, both of which are increased by boiling. The appearance of the urates under the microscope is shown in Fig.

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* The Philadelphia "Medical Times," January 12, 1884.

† The "Medical News" (Philadelphia), May 30, 1885.

Hippuric Acid.—The amount of hippuric acid varies with the diet, and certain unknown conditions of the assimilation. From ten to fifteen grains are excreted in the urine of a healthy adult in twenty-four hours. It is increased in diabetes, and by the use of such fruits as cranberries, blackberries, etc.

The other acids of the aromatic series have but little clinical significance at present.

Kreatinin.—The source of kreatinin is kreatin, one of the products of retrograde changes in muscular tissue. It is an alkaloidal substance of considerable power, but its clinical relations are unknown. The amount excreted in twenty-four hours ranges between eight and twenty grains, it is supposed.

Urine Pigments or Coloring Matters.—These have considerable importance. There are two—urobilin ; indican. The former is derived from *hæmatin*, the coloring matter of the blood, by the action of the bile acids ; the latter from *indol*, a substance produced in the course of the pancreatic digestion of the proteids. Indol, after absorption into the blood, is converted into indican, and is thus excreted by the urine. Urobilin, or a substance corresponding to it, is variously named by different observers. By George Harley it is called *urohæmatin ;* by Heller, *urophain*. It is the most important urine pigment, if not the sole coloring matter. The quantity normal to the urine is increased in certain diseased states—in liver diseases, in fevers, and in certain disorders of the vascular system.

Indican is probably a normal constituent of the urine, but by some it is held to be a pathological product. It is Heller's uroxanthin, and is supposed to be derived from *indol*, a product of pancreatic digestion, and this is converted by the alkaline constituents of the blood into indican. A high temperature of the atmosphere, a meat diet, and other conditions increasing the acidity and density of the urine, raise the proportion of indican above the ordinary level, and in such pathological states as diabetes, cancer of the stomach, obstruction of the intestines, etc., it is so much greater than in health as to have some value as a means of diagnosis.

The presence of indican in the urine may be demonstrated as follows: Warm one drachm of strong nitric or hydrochloric acid in a test-tube; then gently pour on the surface of the acid about the same quantity of urine (which must be free from albumin), and at the point of contact there will form a ring of color—violet, if but little indican is present; and blue, if there be much. The indican may be separated by chloroform. The acid and urine are well incorporated by shaking them up in the test-tube, a little chloride of lime solution added until a greenish tint appears, and then the whole is agitated with two and a half drachms of chloroform. If allowed to stand undisturbed for a time, the chloroform will separate of a violet or bluish color, according to the quantity of indican dissolved in it.

The Mineral Constituents—Phosphates.—The combinations of phosphoric acid with the mineral bases of the body are of two kinds: alkaline and earthy—of potassium and sodium; of calcium and magnesium. The alkaline phosphates are soluble, and do not, therefore, form a sediment; on the other hand, whenever the urine becomes alkaline, the earthy phosphates are precipitated. The acid reaction of normal urine is due to the presence of the acid phosphates of sodium and potassium. Under certain conditions, not well understood, these phosphates are not converted into acid salts, and then the urine becomes neutral in reaction. When the urine is distinctly alkaline on excretion, this reaction is due to the presence, in excess, of the alkaline carbonates, and then the urine effervesces on the addition of an acid.

We owe to Dr. Gee* the demonstration of the remarkable fact, that in cases of ague the phosphoric acid disappears from the urine, or is greatly reduced in amount. In chronic albuminuria the alkaline phosphates are below the normal considerably.

The *earthy phosphates* of systemic origin consist of calcium and magnesium phosphate. The ammoniaco-magnesium phosphate is present in large quantity in certain diseases of the genito-urinary tract, and it is then of local origin, or from the inflamed mucous membrane. Phosphate of lime is found in the urine in two forms: as amorphous granules, and as needle- and star-shaped crystals—the former in much greater quantity. When acid urine is boiled, the phosphates being in excess, a cloud appears, which is dissolved, and the urine made clear by the addition of a few drops of nitric acid. If the urine is alkaline, the phosphates may be sufficient to give it a turbid appearance, or even to make it thick and white. It sometimes happens that an abundant precipitate of the phosphates takes place in the bladder, and the urine last passed is then thick, white, and ropy, and voided with difficulty. This condition of things is alarming to the patient, and may be perplexing to the physician, until, on the addition of nitric acid, the whole sediment disappears. If effervescence is produced by the addition of acid, it may be concluded that the bicarbonates of potassium and sodium are present in excess.

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* "St. Bartholomew's Hospital Reports," vol. viii.

The *ammoniaco-magnesium phosphate*, or *triple phosphate*, is found in the urine when it has been rendered alkaline by ammonia. The ammonia is a product of the decomposition of urea, the mucus present playing the part of a ferment. The crystals of triple phosphate are triangular prisms, and are readily recognized under the microscope. Like the other phosphates, this is dissolved by a mineral acid.

Phosphorus, unoxidized and in combination with organic substances, as lecithin, appears in the urine. The quantity present is estimated by converting it into phosphoric acid by an oxidizing agent. In health the amount excreted by the urine is very minute, but in certain diseases characterized by destructive changes in the brain substance, in phthisis, in pernicious anæmia, etc., it is greater.

The Sulphates.—The sulphates consist of combinations of sulphuric acid with potassium and sodium, and, to a much smaller extent, of calcium. In health the amount of sulphates excreted is largely influenced by diet, especially animal food. In disease they are increased by the inflammatory process, notably pneumonia, meningitis, etc.

The Chlorides.—The chief chloride salt is chloride of sodium, and the quantity of this excreted is much affected by the amount of common salt taken with the food. A portion of the salt eaten is, however, converted into chloride of potassium. From one to two drachms is excreted in twenty-four hours. The test for chlorides is nitrate of silver. The urine to be examined is first acidulated with nitric acid to keep the phosphates dissolved, and then solution of nitrate of silver is added, whereupon an abundant white, curd-like precipitate of chloride of silver falls. This precipitate is not soluble in excess of nitric acid, but is dissolved by ammonia in excess.

It has been ascertained that the chlorides disappear from the urine, or are, at least, greatly reduced in amount, during the progress of an acute inflammation. In pneumonia this change in the quantity of chlorides becomes a prognostic indication of some value, for disappearing as the lung becomes hepatized, they suddenly reappear with the occurrence of the other critical phenomena.

Adventitious Substances.—Diagnostic, prognostic, and therapeutic indications of great value are obtained from the examination of certain adventitious materials which may be present in the urine.

Albumin.—Many tests have been proposed for serum albumin, but it is quite certain that none of them are as accurate as the application of heat. Much depends on the mode of employing it. The following method is the best:

Half fill a medium test-tube with the suspected urine. Heat it to the boiling-point. If the urine is decidedly alkaline in reaction, and no cloudiness appears, add sufficient nitric acid to change the reaction to acid, and then boil again. If now no cloud appears, the urine does not contain albumin. If the urine is neutral or acid in reaction, and on boiling a milky cloudiness becomes manifest, add some nitric acid, drop by drop, by means of a pipette. If the urine clear up, the precipitate is not albumin, but most probably phosphates. When the urine under examination is cloudy from the presence of mucus, it should be filtered ; if from urates, heating will redissolve them, and the urine become clear before it gets cloudy again from coagulation of the albumin.

Heller's *nitric-acid test* is very convenient, but its value is impaired by several possible fallacies. It consists in the coagulation of the albumin by nitric acid. Thirty minims of pure acid are put in a test-tube, and about the same quantity of urine is allowed to flow gently on to the surface of the acid, where, if albumin be present, a whitish coagulum appears, which is not dissipated by heat. Hydrated uric acid, and amorphous urates, may furnish a similar zone to that made by albumin, but these disappear on heating.

Johnson's *picric-acid test* is delicate, and, if confirmed by the action of heat, is certain. It is applied as follows : A saturated solution of picric acid is made by dissolving six to seven grains in an ounce of boiling distilled water, and this solution is poured on some of the suspected urine contained in a test-tube. If the resulting yellowish coagulum is not dissolved by heat, it consists of albumin.

Probably the most sensitive of all the tests for albumin is *Tanret's potassio-mercuric iodide.* It consists of the following ingredients : corrosive chloride of mercury, 20 grains ; iodide of potassium, 50 grains ; acetic acid, 6 drachms ; and sufficient distilled water to make up to 2½ pints. This precipitates albumin, and also peptones, alkaloids, and urates ; but heat dissolves all these, except albumin.

The albumin ordinarily present in urine in disease is serum-albumin —the albumin of the serum of the blood.

Acid-albumin, or syntonin, is occasionally encountered when the urine is highly acid. It is not precipitated by heat until the acidity of the urine is lessened, which may be effected by the careful addition of sodium-carbonate solution. *Alkali-albumin*, or casein, also fails to be precipitated on boiling, unless some drops of acid are added to neutralize the urine.

Paraglobulin is found in albuminous urine associated with serum-albumin, and may indeed be in excess of the latter. Very rarely it appears alone in the urine, and is mistaken for albumin. In cases of albuminuria due to amyloid degeneration, and of scarlatina nephritis, and in temporary albuminuria caused by derangement of the digestive organs, paraglobulin may be in excess. It is separated from serum-albumin by treating the urine with sulphate of magnesia to the point of saturation, when the paraglobulin is cast down.

Peptones are also often found, usually with albumin, but may be present without it. The most delicate and convenient reaction for distinguishing peptones in the urine is that devised by Dr. Randolph, of Philadelphia, and is based on the fact that, when acid mercuric nitrate is added to a solution of iodide of potassium, red mercuric iodide is precipitated; but if peptones or bile be present, the precipitate is yellow. It is necessary, then, to determine whether the bile acids are present by the appropriate tests.

Peptones are present in the urine in cases of acute septic diseases, such as diphtheria, variola, and cerebro-spinal meningitis, and when pus or inflammatory exudates are absorbed, as, for example, in cases of croupous pneumonia, pleuritis, and rheumatic effusions.

In the sections devoted to diseases of the liver, the reactions due to the presence of *bile* pigment and acids are given. It is necessary, however, to say something of *leucin* and *tyrosin*. These are found together in the urine when the liver-structure has undergone extensive injury, as in acute yellow atrophy, in phosphorous poisoning, and in malignant typhus. These substances take the place of urea. For the methods of their detection and appearance under the microscope the reader is referred to works on urinary analysis.

Sugar.—It is now commonly held that sugar is present in very minute quantity in normal urine. Nevertheless, any amount above the merest trace must be regarded as pathological. The tests most in use are three: Trommer's, Fehling's, and fermentation.

As in the application of Trommer's and Fehling's the results may be vitiated by the action of the urates, it is necessary to remove them before applying the reagents. This is accomplished by precipitating them with acetate-of-lead solution, and then filtering. In using Trommer's test, the following method is pursued: To the urine in a test-tube is added a few drops of the solution of cupric sulphate, or a pellet of the same. The sulphate is converted into the oxide by liquor potassæ, which is poured in until the first precipitate is redissolved. If sugar be present, the oxide of copper is reduced to suboxide on warming the liquid, and this reaction is manifest by the appearance of a reddish precipitate. If the reduction of the copper does not take place on warming the liquid, sugar is not present, although on prolonged boiling the reaction may occur.

Fehling's differs from Trommer's test in that the potassio-tartrate of sodium (Rochelle salt) is added to the alkaline copper solution. If the solution be kept for any considerable period, it becomes so changed as to be unreliable, and should be boiled before using as a test, since the copper may be reduced thus. Pellets of copper sulphate and of sodic tartrate of potassium are now prepared separately, and are added to a solution of caustic soda when required for use. Solutions may also be kept separately, and mixed when needed. When a given specimen is to be examined, Fehling's solution is heated to the boiling point, and a drop of the urine is added, when a yellowish precipitate occurs if sugar be present in quantity, or greenish if the proportion of sugar be small; but the yellowish tint is produced by putting in a few more drops of urine.

URÆMIA.

Definition.—The retention in the blood of those excrementitious substances which it is the function of the kidney to remove is known as *uræmia*. Strictly speaking, this term signifies urea in the blood; but, as urea is not the most offending substance, the extractives, and even the potash salts, according to Feltz, and other ingredients of the urine being poisonous when retained in the circulation, the term uræmia has, by common consent, been extended to include the complexus of symptoms due to the presence of the urinary constituents in the blood.

Pathogeny.—The retention of the urinary constituents may be due to insufficient secretion or to imperfect excretion; in the former, the materials which go to make up the urine are not separated from the blood; in the latter, after the urine has been duly formed, its excretion is prevented, and resorption into the blood occurs. In the various forms of albuminuria, uræmia is due to insufficient secretion, and imperfect excretion results from obstructive disease of the ureters, of the bladder, of the urethra, and from exterior pressure, as in the case of tumors encroaching on the urinary passages.

The diminution of the urine, which is concerned in the production of uræmia, consists in the lessened excretion of the solids. The urinary water may, in respect to bulk, be fully up to, even exceed, the normal, and yet symptoms of uræmia occur, because of the insufficient depuration of the urinary excrement. Great differences of opinion have existed, since the time of Bright's discovery, in regard to the particular urinary constituent causing the secondary disturbance. Christison was the first to express the idea of a connection between certain nervous phenomena and the condition of albuminuria, and he referred these phenomena to blood-poisoning caused by the retention of urea, but he also held that there was an excessive excretion of the coloring matter to which the disturbance was in part due. Hammond, and subsequently Richardson, maintained that the retention of urea is the real cause. Frerichs then brought forward his carbonate-of-ammonia theory. He showed, what subsequent observers have confirmed, that urea itself injected into the veins is not injurious, and hence suggested that the real poisonous principle is carbonate of ammonia, since convulsions are produced when this substance is thrown into the veins, and it is found in the blood in uræmia. Against Frerichs's theory were opposed the insuperable objections that ammonia is a natural constituent of the blood, and that not more of it is found in the blood of animals from which the kidneys have been removed than in the normal state. Treitz enlarged Frerichs's ammonia theory so that it included more facts, but it is not satisfactory. Treitz held that carbonate of ammonia is the toxic agent, but maintained that urea, excreted by the mucous membrane of the intestinal canal, is there decomposed, and diffused thence into thê blood. Schottin next proposed that uræmia is due to the retained extractives, creatine, creatinine, etc., which are products of tissue metamorphosis, that later in the process of hystolytic transformations are converted into urea. Although these substances may be noxious when retained, the theory which restricts the phenomena of uræmia to their retention is insufficient in scope. Traube has also propounded a theory, in which doubtless there is a measure of truth, since it is supported by the only lesion which is really referable to the condition of uræmic intoxication, namely, œdema of the brain. By reason of the watery state of the blood, transudations of serum take place through the cerebral capillaries, producing œdema of the brain. With the increase of fluid in the perivascular lymph-spaces, more and more pressure is exerted on the intra-cranial vessels, so that ultimately sufficient anæmia is produced to cause convulsions. The existence of more or less œdema of the brain is doubtless true, and this facilitates the action of the toxic agents, while it is in part a result of their action. The last theory to be referred to is that of Feltz and Ritter, which ascribes uræmia to the toxic action of the potash salts of the urine, retained in the blood. Unquestionably the nervous and other symptoms of uræmia may be produced by injecting the potash salts into the veins. But, while this statement is admitted, it may also be affirmed that not to one, but to all of the retained constituents of the urine possessed of toxic activity, may be referred the phenomena of uræmia. When there are several capable of producing disturbance of this kind, it were wiser to include them all, and refer the condition of uræmia to the retention of the toxic urinary constituents.

Symptoms.—Uræmia may be *acute* or *chronic:* the former occurring in those renal affections characterized by a sudden great diminution or suppression of the urine; the latter belonging to the chronic affections, such as chronic interstitial nephritis, in which the quantity of urinary solids is slowly reduced. Acute uræmia consists in sudden violent headache, vertigo, disordered vision, twitchings of the facial and other muscles, followed by general convulsions of an epileptiform character. The convulsions may recur every few minutes or hours, and in the interval there may be coma, the condition of insensibility persisting until death, or until the renal functions or vicarious discharges are reëstablished. The convulsions may exist without loss of consciousness, and in some cases assume a tetanic rather than an epileptic type. The acute form may also occur without convulsions, the patient passing from a state of somnolence into profound coma, in which the face is pallid; the pupils are dilated, but react to a strong light; the pulse is slow; the respirations are shallow, often irregular (Cheyne-Stokes), and sometimes stertorous; and there is general muscular resolution, but not localized paralysis. From this condition the patient may emerge, some hebetude of mind and diminution in general and special sensibility remaining for some minutes or hours, and then recurrences of the coma and periods of improvement until the end. In other cases this comatose form of acute uræmia is varied by attacks of a convulsive character or by a mild delirium.

Acute uræmia assumes still other forms, which, however, are extremely rare. There is a delirious form, in which the mental trouble assumes the shape of a tranquil delirium, less often of acute mania, and is preceded by headache, disorders of vision, by a listless apathy, dullness of apprehension, and weakness of will. There is a dyspnœic form, characterized by the sudden onset of intense dyspnœa without any change in the respiratory organs, but accompanied by a hoarse voice and sibilant inspiration, and terminating in a fatal coma.

The chronic form of uræmia is characterized at the outset by symptoms referable to the digestive organs—dyspepsia, nausea, and vomiting, without apparent cause. With these stomachal symptoms are associated headache, vertigo, and dimness of vision. The headache is very intense and persistent, and is often frontal, and accompanied by a band-like feeling. Early changes occur in the retina—a form of retinitis, known as retinitis albuminurica—which is often diagnosticated by the ophthalmologist before the urine has been examined. Drowsiness is soon experienced. At first the patient falls asleep early in the evening, but after a time he dozes at all times in the day whenever his attention is not attracted by objects of interest. All of his special senses become dull, but vision suffers chiefly, hemiopia and diplopia being the most usual forms of derangement. Meanwhile the nausea and vomiting increase, the headache becomes more severe, vision grows more obscure, and presently muscular cramps occur at night, and twitchings of the facial muscles are observed at various times. Then develop the symptoms already described under the acute form.

Course, Duration, and Termination.—The progress made by a case of uræmia depends on the changes occurring in the kidneys. In a fatal case of acute Bright's disease, the duration of the uræmia in the severe form will not be greater than from three to five days. There are rapid cases, which terminate fatally in a few hours. The chronic form may occupy weeks or months. In the acute forms of Bright's disease opinions in regard to the termination of uræmia must be expressed with caution, since, under the proper treatment, very formidable symptoms may disappear and health be restored. This statement is especially true of scarlatinal and pregnancy albuminuria. In chronic interstitial nephritis, in amyloid disease, and other fatal diseases, the appearance of uræmia is significant of a fatal termination. The chronic form of uræmia is, therefore, more serious than the acute.

Diagnosis.—The state of the urinary secretion is the first element in making a diagnosis. Is the secretion suppressed? Does the urine contain albumen or morphotic constituents, indicating structural changes in the kidneys? Albumen and casts wanting, does an analysis reveal a notable diminution in the solids of the urine? As regards the cerebral symptoms of uræmia, two points are especially significant: the absence of motor paralysis and of fever, which prove that meningitis, hæmorrhage, encephalitis, etc., are not the causes of the disturbance. That opium, belladonna, strychnine, and other narcotic poisons are not concerned in producing the phenomena of uræmia, is evident from the history of the case and the state of the urine. In every case of convulsions or sudden insensibility, not explainable by known conditions, if the urine has not been preserved, the catheter should be promptly used. If unconsciousness has been produced by a narcotic poison, the nature of it may sometimes be determined by injecting some urine under the skin of an animal.

Treatment.—As uræmia is due to the retention in the blood of excrementitious matters which ought to have been separated by the kidneys, obviously some mode of vicarious relief becomes necessary. By the skin and intestinal canal, excretion may be in considerable part effected. By the vapor-bath, and pilocarpine subcutaneously, the skin may be efficiently acted on. When the heart is weak, pilocarpine must be cautiously used; also, in case there be much bronchial mucus and weak respiratory muscles, this agent may not be safe. When no contraindication exists to its employment, there can be no question in regard to the good effects produced by it. As urea and the product of its decomposition—carbonate of ammonium—is eliminated by the gastro-intestinal mucous membrane, when excretion by the kidneys is stopped, we may imitate the method of nature, and free the blood of excrementitious matters by purging. For this purpose the compound jalap powder, podophyllin, calomel (cautiously), may be used. The kidneys may be stimulated to better action, when not too far gone, by the administration of saline diuretics, infusion of digitalis, etc. When convulsions occur, more prompt means of relief become necessary. The inhalation of chloroform is an excellent expedient, and is free from danger, if judiciously conducted. The hypodermatic injection of morphine, as proposed by Loomis, is a valuable expedient, if it may seem rather heroical. He advises the injection at once of a half-grain of morphine for an adult, and this is repeated two or three times if necessary. The author has seen cases yield to this which continued under chloroform inhalations. This method is adapted to the cases of acute uræmia. Morphine thus administered seems to antagonize the condition of the cerebral vessels induced by uræmia, removes the œdema of the brain, and starts the kidneys secreting again.

CONGESTION OF THE KIDNEYS—ACTIVE.

Definition. — Hyperæmia of the kidneys signifies an increased amount of blood in the organs. The hyperæmia may be in the arterial supply—*active congestion*, or in the venous supply—*passive congestion.*

Causes.—*Active congestion* is usually caused by some irritating substance which is eliminated by the urine. Various medicinal agents, containing an essential oil, or a camphor, as copaiba, cubebs, eucalyptol, etc., excite irritation in the kidneys, as these substances pass through in the process of elimination. Turpentine and cantharides are among the most active of these agents, and more frequently cause acute congestion than any other. A mustard-plaster may also have the same effect, due doubtless to the absorption and elimination of the oil of mustard. An extensive burn, a counter-irritant affecting a considerable extent of surface, and possibly other injuries or impressions on peripheral nerves, may induce a reflex paresis of the arterioles of the kidneys.

Symptoms.—More or less pain, sometimes very acute pain, is felt in the region of the kidneys, and extends downward along the course of the ureters, into the hips, through the bladder, which becomes very irritable, and into the testicles and penis. There is present an incessant and very pressing desire to pass water, which is high-colored, and rather scanty each emission, although in the aggregate up to the normal. The urine may contain blood, or but a few red globules, or simply fibrin and casts, some cells of renal epithelium and albumin. If the action of the cause continue, the state of hyperæmia will pass over into some of the forms of inflammation. The author is convinced that the persistent use of copaiba has kept up an hyperæmia, out of which has developed the chronic form of Bright's disease. If the agent producing the hyperæmia is withdrawn, irritation subsides in two or three days, and health is restored.

The only *treatment* required in the mildest cases is to withdraw the irritating agent, to dilute the urine by the free administration of lemonade, or Vichy water, or Bethesda water. If there are decided irritability of the bladder and much pain, relief is quickly afforded by the administration of two or three grains of camphor every four hours, or still more promptly and efficiently by the hypodermatic injection of one twelfth of a grain of morphine, or by the stomach administration of one sixth to one fourth of a grain.

CONGESTION OF THE KIDNEYS—PASSIVE.

Causes.—*Passive congestion* of the kidneys is caused by venous stasis. The chief lesions inducing venous stasis are obstruction and regurgitation of the mitral orifice, obstructive diseases of the lungs, obstruction and regurgitation at the tricuspid orifice, compression of the ascending vena cava above the renal veins, and thrombosis of the renal veins.

Pathological Anatomy.—The vessels are abnormally full, and hence the organ is larger, and more blood flows out on section. As there is a moister state of the organ, owing to mechanical effusion from the swollen veins, the capsule is easily detached. The parenchyma of the organ is darker, having a bluish aspect ; it is moist and smooth ; the glomeruli are not swollen and congested, but the vessels of the convoluted tubes are distended. The stellate vessels of the surface can be traced with the eye into the anastomoses of the interfascicular veins, and the vessels of the vasa recta are recognized as dark reddish striations (Rindfleisch). If hyperæmia becomes chronic, the over-supply of venous blood leads to important nutritional alterations—to hyperplasia of the connective tissue—and hence the whole organ increases in size, firmness, and weight.

Symptoms.—In cases of passive congestion of the kidneys, the central disorder quite masks the changes occurring in the kidneys. When dropsy occurs, attention is directed to the state of the urinary secretion, but previously no symptoms had arisen indicating that the kidney was suffering. Besides the venous stasis and increased pressure in the venous system, the disturbance in the urinary function is in part due to the diminished pressure in the arterial system. The urine is scanty, dark in color, and acid in reaction. On standing, a very abundant deposit of urates takes place, and the urine becomes thick. The specific gravity is increased in the ratio of the decrease in the urinary water, and is 1025 to 1035, but it is also high because of the quantity of solids, uric acid, notably of urea, which may rise to five per cent., or higher. An important change now is apparent in the composition of the urine—it contains more or less albumin, but not often any considerable amount. If such urine, thick and dark, is placed in a test-tube and gently heated, it will soon clear up, except some fine particles, but gradually, the heat continued, the clear urine will become milky, from the coagulation of albumin. The urates dissolve at the temperature below the coagulating point of albumin. On microscopic examination the morphotic elements present in the urine consist of a few red blood-globules, some tubular epithelium, and a few delicate, transparent casts. The amount of albumin present in such urine does not often exceed one per cent.

Course, Duration, and Termination.—The kidney complication in cardiac and pulmonary obstructive disease follows the fortunes of the central lesion. When the cardiac lesion is compensated, and the pressure rises in the arterial and falls in the venous system, the congestion of the veins and the ischæmia of the arteries of the kidneys will cease—the urinary water will increase, and the albumin will disappear. If, however, the central lesions be permanent, the condition of the kidney will grow worse, the albumin increase, and, after a time, the specific gravity will fall. Cerebral symptoms do not arise from venous congestion of the kidney, because the tubular epithelium remains sound and whole, and therefore equal to its function of excreting excrementitious materials. Death may occur from some intercurrent malady, or the patient die exhausted from the persistent dropsical accumulation.

Treatment.—The management of passive congestion of the kidneys is that of the central lesion. It includes the use of digitalis, quinine, and iron, of hydragogue cathartics, of warm baths, vapor-baths, and pilocarpus, caffeine, convallaria, nitro-glycerine, of diuretics, etc. The condition of the kidneys is improved by those remedies which affect the heart trouble favorably. The account already given of the treatment of cardiac disease with dropsy is equally applicable here.

ACUTE PARENCHYMATOUS OR CROUPOUS NEPHRITIS.

Definition.—Under the head of "Bright's Disease" there are included several acute and chronic affections of the kidneys, which agree in the one important characteristic of the urine containing albumin. According to many authorities, acute parenchymatous nephritis is the first stage of Bright's disease: it is "the large, white kidney," "the large, smooth kidney" of English authors, and corresponds to Johnson's "acute desquamative nephritis." Although Charcot adopts the term "parenchymatous nephritis," he holds that we are not yet prepared to name it accurately.* By Bartels it is designated "acute parenchymatous nephritis"; by Millard, "croupous nephritis"; the latter is the more happy term.

Causes.—To this form of nephritis youths are more liable than the aged. An exception to this exists in infants, and the liability continues till middle life, and, indeed, though greatly diminished, does not entirely cease after this period. Heredity appears to have an influence, although the facts are not numerous. Type of constitution seems very important among the causes. The pale, light-haired, full but flabby subjects of the albuminous type seem to have a special susceptibility to this form of nephritis. Those substances which cause active hyperæmia of the kidneys, as cantharides, turpentine, copaiba, etc., will induce inflammation of these organs, if they continue in action for a sufficient time. Scarlatina is probably the most common cause. It is not the character of the epidemic, nor the severity of the attack itself, which wholly determines the changes in the kidneys, for the mildest epidemics and the least pronounced cases may be remarkable for the extent of the renal complication; yet, if the epidemic have a malignant aspect, there will be more formidable cases of nephritis. As not all cases of scarlatina are accompanied by the renal disease, there must be some inherent bodily condition, or peculiarity in the structure of the kidneys, to account for the result. The same is true of diphtheria, in which an inflammation of the kidneys occurs in a proportion of the cases. But in diphtheria there seems to be a relation between the severity of the systemic poisoning and the occurrence of the renal complication. Oertel maintains that the disease of the kidneys is due to the transference to these organs of "bacterian colonies" and their subsequent multiplication. In diphtheria, more than in scarlet fever, there may be albumin in the urine, without recognizable changes in the structure of the kidneys. In analogous morbid states acute parenchymatous nephritis may be produced. These are typhoid, erysipelas, malignant pustule, etc.—diseases due to the reception and development of some specific infective material which, eliminated by the kidneys, excites inflammation in passing through these organs. The skin and kidneys stand in intimate functional relation to each other, and when one is inactive the other may act vicariously in its stead. This physiological fact has a corresponding pathological relation. Acute nephritis may be excited by exposure of the body to cold when the skin is warm and perspiring. The sudden arrest of the skin secretion throws a greatly increased labor on the kidneys; their vessels dilate, and an acute hyperæmia prepares the way for inflammation. Pregnancy is a cause of acute parenchymatous nephritis. Usually, but not invariably, it is the first pregnancy, and it is more common in twin pregnancies. It occurs in the thin, in the robust and plethoric, in those of low and high degree, and under the most varying conditions. Having occurred in one pregnancy it may happen again, and not unfrequently becomes a permanent malady pursuing a course independently of pregnancy. No satisfactory explanation has thus far been offered. That it occurs not more frequently than one time in one hundred and fifty pregnancies renders it probable that there must exist a renal or constitutional disposition which pregnancy excites into activity.

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* On "Bright's Disease," translated by Millard. New York: William Wood & Co.

Pathological Anatomy.—The changes in the structure of the kidney in acute parenchymatous nephritis are much disputed. To render clear the form of the disease under consideration, it may be repeated that it is the large, pale, and smooth kidney of the English writers. It is increased in size, so that it may reach twice its normal weight and volume; the cortex is pale, grayish-white, or a dull white; it is smooth, because when the capsule is stripped off there are no pits or elevations as occur in the contracted kidney, and its texture is rather soft. There is but little hyperæmia of the cortex; here and there dark-red points are seen, or punctiform extravasations; but the pyramids are deeply congested, bluish red, or brighter red, and contrast strongly with the pale gray of the cortex. In other cases, according to Bartels, the cortex may not be so pale, may be reddish gray in consequence of a considerable hyperæmia, and there may be between this amount of congestion and the dead-white a great deal of variation.

The changes ascertained on microscopical examination are found "localized almost exclusively in the convoluted tubes" (Charcot), and consist in cloudy swelling of the epithelium, which remains *in situ*. The change in the appearance of the epithelium—the cloudiness—is due to the deposit of fine granulations, and in such large numbers that the lumen of the canal is almost closed by the distention of the epithelial cells. The ends of the tubules are also sometimes blocked by the deposit of fibrin-plugs. The convoluted tubes also become dilated and varicose by reason of changes taking place in the proper tunics of these tubes. The appearance of the kidney thus affected may be changed by localized or extensive fatty metamorphosis—by fatty change limited to a few tubes here and there, or by a general fatty change. When thus altered the color becomes yellowish, and, if localized, gives to the organ a granular appearance, and hence the name applied to it by Johnson as the *fatty granular kidney;* if general, it becomes the *large fatty kidney*. It has been much disputed whether the large, smooth kidney ever undergoes an atrophic change. It is held by Charcot that in very rare instances an atrophy may be effected by the liquefaction and disappearance of the fatty epithelium and the subsequent collapse of the tubules.

Symptoms.—When parenchymatous nephritis occurs during the course of scarlet fever, diphtheria, and other febrile diseases, the symptoms are modified in various respects. Two modes of onset are described when the disease occurs independently—one sudden, with high fever, aching pains in the lumbar region; the other slow, obscure, and with little disturbance. The first variety usually results from taking cold; the patient, while heated and perspiring, plunges into cold water or lies upon the damp ground, and in a short time—twelve to twenty-four hours—has some chilliness, even a rigor, followed by high fever, intense headache, pains in the lumbar region and through the limbs, nausea, vomiting, and anorexia. The symptoms which attract attention to the kidneys in either mode of onset are the changes in the character of the urine. In some cases the first symptom referable to the urinary organs is an extremely irritable state of the bladder, frequent desire to micturate; a few drops only, and these it may be bloody, can be passed. This symptom does not last long, and is not common. Usually there are observed changes in the quantity of the urine, the amount passed in twenty-four hours being variously reduced from forty ounces, the normal quantity for an adult, to twenty, ten, even five ounces, and at the same time important new constituents appear in the secretion. There may occur entire suppression, when the most formidable symptoms will arise, and death result in a few days. The urine at the onset often contains blood, when it presents various appearances according to the quantity present: it may have a faint, smoky tinge, or with this there may be an admixture of a reddish hue, or it may be distinctly reddish without the smoky hue, or it may be dark, reddish-brown, almost black. When permitted to stand, a quantity of urates fall, and with them various morphotic constituents, chiefly blood-corpuscles, entire or disintegrated. The quantity of urea, as compared with the amount of urine, is much less than normal; uric acid is not less, but the saline constituents are reduced. The gross amount of solid constituents is, therefore, below the standard of health. The reaction of the urine is acid and the specific gravity is high, often reaching 1030, but this result is due to the diminished amount of water, since the solids in the aggregate are below normal. In the further progress of the case, as the amount of water increases, the specific gravity falls; but there is an increase in the solids and in the urea in the aggregate, although the quantity of each is small in any single specimen of the urine. The decline in specific gravity may be from 1030 to 1005. With the diminution of specific gravity or increase of water the acid diminishes, the urine becoming very faintly acid or neutral. The most characteristic condition as regards the urine is the presence of albumin, in this affection ranging from distinct traces to three per cent. The albumin may be absent at the initial period, but only for a brief period, the aggregate amount of the urine being very small. Besides albumin and blood-globules, perfect and disintegrated, there are present casts of the tubules, of coagulated blood, and pale, transparent, hyaline casts, with an occasional epithelial cell adherent. The pale casts are usually few in number, but in the progress of the case they are supplanted by large hyaline casts and numerous large

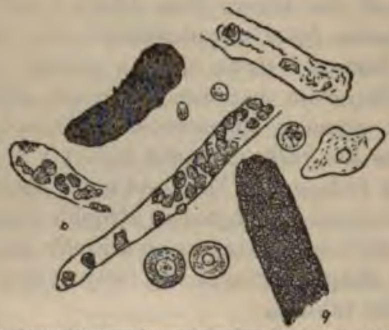

FIG. 37.—Casts of Acute Parenchymatous Nephritis. (Beale.)

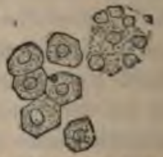

FIG. 38.—Epithelium from Convoluted Tubes. (Beale.)

granular casts. Usually, also, the sediment contains epithelial cells cast off from the tubes and granules in great numbers. Very often it is not until œdema of the ankles and feet appears that attention is called to the state of the urine, when it is found to be scanty. In consequence of the diminution in the amount of water separated by the kidneys, the condition of the blood and the rate of absorption, especially, the cellular tissue becomes œdematous ; if the patient is up, the water settles in the feet and legs ; if recumbent, it accumulates in the lumbar region and hips, and may first, or coincidently with its appearance elsewhere, manifest itself in the eyelids. Puffiness of the face, with a peculiar pallor of the skin, and broadening of the bridge of the nose, while the eyelids are swollen, present a striking appearance which can hardly fail to be observed, and may be the first indication of the œdema. The effusion extends, the subcutaneous areolar tissue becomes universally filled, and the great serous cavities are ultimately distended to their utmost.

The retention in the blood of the excrementitious substances in health discharged by the kidneys has a disastrous effect. The nervous system is poisoned, convulsions (eclampsia) occur and vary in severity, from twitching of the muscles of the face and of the extensors of the forearms to general convulsions involving loss of consciousness and clonic spasms of all the voluntary muscular system. The appetite is lost, and there are usually nausea and protracted vomiting, and sometimes there is very troublesome diarrhœa. The loss of albumin and of blood and the poisoning of the blood by retained excrementitious matters soon lower very seriously the nutrition of the body. Vision is impaired, both in consequence of simple anæmia of the retina and of the changes of albuminuric retinitis.

Course, Duration, and Termination.—Those cases occurring spontaneously are more acute in character, accompanied by fever and disorders of micturition, which attract attention to the kidneys. The fever does not continue longer than a few days. If there is complete suppression, the case may terminate fatally within a week. If, as is usual, the development is slower and the urine is greatly diminished in quantity, the amount of the dropsy will depend on the reduction of urine for a lengthened period. The promptness with which œdema appears is determined by the scantiness of the urine, so that well-developed dropsy may be produced in a week. When the cellular tissue and the cavities are filled with fluid, the duration of the case depends on the degree in which the kidneys can be made to functionate, for, although temporary improvement and alleviations may result from vicarious discharge of the urinary functions, results obtained in this way are not permanent. This form of nephritis is not nearly so fatal as the other forms ; indeed, the percentage of recoveries is quite large. When this disease occurs in scarlatina, it modifies the course of the latter materially, and prolongs its duration. Death may ensue in convulsions, or result from exhaustion in consequence of the protracted anæmia, and the gastro-intestinal disturbance, which prevents the retention and assimilation of food. Recovery may ensue after several weeks of dropsy, vomiting, and diarrhœa, interspersed with eclampsia, the convalescence being very slow. Three months or more may be occupied in the return to health.

The Acute Parenchymatous Nephritis of Pregnancy.—There are points connected with this disease requiring special consideration in respect to its course and terminations. It is usually considered due to two factors—to the relatively poor quality of blood of pregnant women, and to the pressure of the enlarging uterus on the renal veins, causing passive congestion. As Bartels shows, the renal veins occupy a position which secures them against pressure, and, as so large a proportion of pregnant women escape the complication of albuminuria, it can hardly be due to either or both of the factors to which it is usually ascribed. There must be some special predisposition, and as the condition of the kidney is precisely the same as in the acute parenchymatous nephritis, and as it not unfrequently assumes the chronic form, pregnancy is merely an exciting cause. The change in the kidneys may take place in the early months of pregnancy, when visual disturbances, dropsy, and miscarriage will ensue, or later, when to the visual disturbances and dropsy must be added eclampsia. Œdema of the face and limbs and frequent micturition are often the first symptoms, but, in the author's experience, visual disorders, especially hemiopia, double vision, and amblyopia, are very frequently the first departure from health.* Again, persistent huskiness of the voice may be the first indication. In other cases no symptoms are felt but disorders of digestion, and, as they are like those of the first months of pregnancy, little attention is paid to them, or there may be persistent headache with vertigo. Sometimes the first symptom to attract attention is an attack of convulsions, the health being apparently good. The urine usually contains an excessive quantity of albumin. The œdema is usually not great. The important point in these cases is the violence and acuteness of the uræmia, whether manifest in the form of convulsions or maniacal excitement. The relative frequency of eclampsia in proportion to the whole number of cases of albuminuria is about one fourth, and of those attacked by eclampsia about one third die. The symptoms usually quickly subside on abortion or delivery, but a considerable proportion become chronic and prove fatal in subsequent pregnancies.†

Treatment.—As the kidneys are in an irritated state, all stimulants to them should be avoided. To give them rest, vicarious functions need to be stimulated to the highest activity—notably the skin and intestinal mucous membrane. When the symptoms are urgent, the skin may be excited by pilocarpine nitrate ($\frac{1}{12}$ to $\frac{1}{8}$ gr. for an adult), or by the vapor-bath or warm pack. As Barker, of New York, has recently shown, pilocarpine must be used with caution in these cases on account of its depressing effect on the heart. Those purgatives are used that produce free watery evacuations. If the stomach is very irritable and the symptoms not urgent, small doses of calomel ($\frac{1}{6}$ grain), frequently repeated, act extremely well. In acute uræmia, the most active cathartics are required—as elaterium, croton-oil, gamboge, etc.—since it is necessary to procure abundant watery evacuations. If the case does not require immediate active interference, the compound jalap powder is probably the most generally useful of the purgatives in this disease. It is best administered in the early morning, so that the disturbance produced by it may be ended before the time for the administration of the other remedies directed during the day. To relieve the kidneys of congestion, and to remove obstructions from the tubules, diluents must be freely used. The most important diluents are milk and cream-of-tartar solution. If the stomach is irritable, milk may be given with lime-water, one fourth to one third of the latter. Infusion of digitalis may be given with cream-of-tartar solution, or alone ; but it is more effective in combination. If the stomach will not bear digitalis, it acts surprisingly well in the form of a poultice applied to the back or abdomen. Nitro-glycerine is very useful : it lessens the congestion of the kidneys, and diminishes the amount of albumin present in the urine. It is best given in the form of the centesimal (one per cent.) solution, commencing with one drop every four hours and adding a drop at each dose until the characteristic effects are produced.

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* See "Die Albuminurie in ihren ophthalmoskopischen Erscheinungen," by Dr. Hugo Magnus, in which the changes in the retina wrought by albuminuria are well depicted.

† Elliot, "Obstetric Clinic," chapter iii, New York, 1868.

If eclampsia occur, what treatment is most effective? If the subject is plethoric, the superficial veins full, the conjunctiva injected, bleeding, by venesection, may be practiced with advantage. Chloroform, by inhalation, can be used to abate the violence of the symptoms, but as soon as possible a hypodermatic injection of morphine should be given according to the method of Dr. Loomis, of New York, who has shown that large doses are remarkably effective in arresting the convulsions of uræmia. Half a grain of morphine can be given at once, and it may be repeated in two or three hours, if necessary, until two grains have been taken. He shows that, if the first large dose is without effect, other doses should be administered fearlessly until the desired effect is produced. Warm baths and active purgatives are indicated, and must be energetically used. Excellent results have been obtained by the use of chloral by the stomach (gr. xv to gr. xlv) : it is even more effective by the rectum. Bromide of potassium may be given in full doses, with or without chloral, by the stomach or rectum, according to the condition of affairs. The same principles hold good in the treatment of the puerperal mania arising from uræmic intoxication.

CHRONIC PARENCHYMATOUS OR CROUPOUS NEPHRITIS.

Causes.—It is comparatively rare for the chronic form of parenchymatous nephritis to succeed to the acute. It is a disease of youth, and is rare after forty. It arises from those causes which depress more or less permanently the vital forces, as syphilis, chronic malarial poisoning, protracted suppuration, chronic alcoholismus, chronic mercurialismus, and other chronic poisoning by metals, etc.

Pathological Anatomy.—To this form of diseased kidney is the term large, pale, or white, smooth kidney, especially applicable. One or both may be affected. The capsule is thin because of prolonged stretching, and, when divided, flies apart and is easily detached. The cortex is a dull, rather yellowish-white color, and is anæmic, while the pyramids are full of distended vessels and are dark red. The enlargement is due chiefly to an increased thickness of the cortical part. The epithelial lining of the tubules is not simply affected with "cloudy swelling," as in the acute form, but has undergone important changes—has been either detached, or is far advanced in fatty degeneration, the cells being filled with fat-globules. The tubules contain a detritus, the product of the destruction of the epithelium, and consists largely of oil-globules, and they also are seen to be blocked in places by large casts. The intertubular matrix is also greatly thickened—a change due to hyperplasia of the connective-tissue elements, to the migration of the white corpuscles and their subsequent multiplication and fatty transformation, and to a quantity of fluid exudation, the product of the increased pressure in the veins. The Malpighian tufts and arteries are sometimes affected, according to Bartels, with the amyloid change in cases arising from chronic suppuration.* Undoubtedly, many tubules are rendered entirely and permanently useless, but restoration may take place when extensive changes have occurred in the kidneys. But, when the changes are too far advanced to permit recovery, the increase in the intertubular connective tissue and its subsequent contraction bring about an atrophic degeneration.

Symptoms.—The approach of this form of kidney-disease is insidious. There is some decline in strength, the body is more easily fatigued, the mind is rather sluggish, and the appetite is poor. A condition of anæmia is evident, and the face has an earthy or fawn color, but it is not until œdema appears about the eyelids and ankles that advice is sought and the real nature of the case made apparent. The accumulation of fluid now proceeds rapidly, and in a short time the whole body is greatly swollen. The cellular tissue, the penis, and scrotum are immensely distended, and afterward the cavities fill up to their utmost capacity, and death may be soon caused by œdema of the lungs or paralysis of the heart. The dropsy in this form of nephritis assumes much greater proportion than that of the acute, or indeed of any form of nephritis. As the accumulation of fluid increases, the amount of urine discharged diminishes, but the urine falls off with the beginning of the renal lesions, although the change is not enough to attract attention. When the disease attains its maximum, the quantity of urine passed in twenty-four hours becomes exceedingly small, and may not exceed four ounces, but there is considerable fluctuation, due to the variations in the amount of water. The urine has a darkish, smoky-looking color, which deepens as the quantity lessens. As the urine cools, it becomes thick with urates, epithelium, casts, etc. The sediment, which falls in great quantity, is composed of urates, uric acid, casts, white blood-globules, and granular detritus. The casts at first consist of pale, delicate hyaline cylinders, dotted here and there with oil drops or granules, either long, narrow, and curved, or broad and shorter. The casts change in character with the progress of the case, becoming more granular, fatty, and the broad replacing the narrow casts. The specific gravity of the urine changes with the variations in the quantity of urinary water, rising to 1035, even 1040, when the amount of urine discharged is very small. If, from any cause, there is a considerable increase in the quantity of urine, the specific gravity falls correspondingly, and below the normal. Albumin is always present, but not in very great quantity, and fluctuates in amount with the variations in the specific gravity. The same fact is true of urea, which, while constantly and absolutely below the normal, varies with the changes in the specific gravity of the urine. The uric acid is increased, and probably in the ratio of the diminution of the urea.

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* Rindfleisch, while admitting the existence of amyloid change, regards it as "infrequent." (*Op. cit.*)

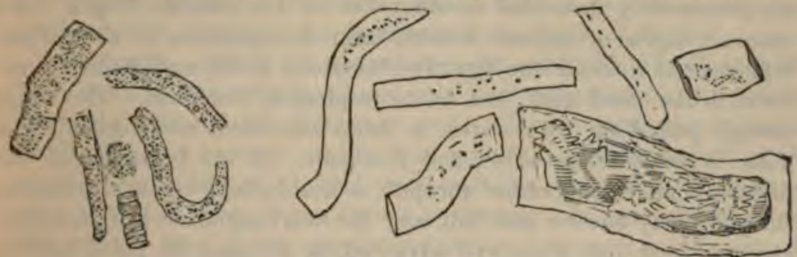
FIG. 39.—Casts. (Beale.)

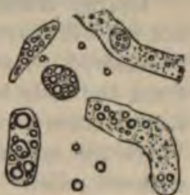
FIG. 40.—Casts becoming fatty.

When the dropsical accumulation has reached the maximum, the fluid is not limited to the subcutaneous tissue and the cavities. The mucous membranes become similarly affected. An early symptom may be a husky, even toneless voice, and dangerous laryngeal stenosis, from œdema of the glottis. The lungs become more or less œdematous at the height of the disease, and life may be terminated by the accumulation of fluid in the lungs. The gastro-intestinal mucous membrane is also dropsical, and the epithelium, swollen, sodden, and degenerating, is cast off in large quantity. The result is vomiting of a quantity of serous fluid and profuse serous evacuations from the bowels, not only exhausting in themselves, but causing, ultimately, greater depression by interfering with digestion and the assimilation of food. The external integument is similarly affected. The epidermis is sodden and detached; the skin cracks in places, permitting the water to drain through; and the true skin, irritated and exposed, becomes exceedingly painful. This process takes place especially where the enormously distended scrotum lies on the swollen thighs. An extreme degree of anæmia results, from the operation of the various influences at work, in the digestive functions, in the assimilative functions, in the blood itself, and in the respiratory functions. The body, though puffed up with water, is thin, emaciated, and feeble. The pulse is small, compressible, and frequent. At the beginning of the disease, commencing rather abruptly in healthy and vigorous subjects, the pulse may be slow and full, and the heart-sounds sharply accentuated and loud, but, when well advanced, in all cases the pulse has the characteristics just mentioned, and the heart-sounds are feeble and obscure. When œdema of the lungs takes place, the respiration becomes embarrassed; but, if large serous accumulations occur in the pleural cavities and in the pericardium, the breathing becomes very difficult, the patient is unable to lie down, and is tormented by a feeling of impending suffocation. Uræmia does not occur so frequently in the chronic as in the acute form of the disease, but amaurosis, muscular twitching, and partial and general convulsions do now and then take place.

Course, Duration, and Termination.—Commencing insidiously, it is not until dropsical symptoms are manifest that the nature of the case is declared. Rarely does the disease come on with boisterous symptoms, the body becoming rapidly distended. When the œdema is observed, there is no long interval in any case until the dropsy is general. When the maximum distention is reached, life can not long continue without relief. Dropsy, however, does not appear at once in every case—albuminuria may exist for months without any effusion, but, when this is the case, there may properly be a suspicion that an error of diagnosis has been committed. In favorable cases the dropsy will not be so great, and the kidneys will manifest a disposition to activity, and will respond to the action of medicines. Those are unfavorable cases in which the dropsical accumulation is extreme, and the kidneys are sluggish, but little urine passing, and in which these organs can not be induced to act efficiently. When there is pronounced dropsy, if the urine increases and the effusion diminishes, a year or more must be expected to pass before recovery can ensue. A complete recovery is by no means a rare event. Usually, when the dropsy disappears, and convalescence is apparently established, there are yet albumin and casts in the urine. If this is the case, the recovery is not real: there may be a slow return of flesh, the cachexia may diminish, and the strength improve, but a return of the dropsy may be confidently expected. Usually, when the albumin persists in the urine, the health is not restored when the dropsy disappears, but the body continues emaciated, and the pallor and anæmia remain. Death may be due to some intercurrent malady—to an acute serous inflammation, to a low grade of pneumonia, etc.; or the patient may be worn out and die by exhaustion; or death may be due to uræmic coma. That the last-named accident does not occur more frequently is probably due to the fact that the excrementitious urinary substances are contained in the fluids of dropsy.

Diagnosis.—When the symptoms occur suddenly, there is feverishness, the urine contains blood and pale casts, and there is pain in the back, the form of the disease is acute. If the symptoms come on slowly, there is no fever, no blood or epithelial cells are present in the urine, the quantity of albumin small and the specific gravity high, or over 1030, the form of the disease is chronic. In contracted kidney, the urine is pale, of low specific gravity, and contains waxy casts; in chronic parenchymatous nephritis the urine is dark, of high specific gravity, and contains abundant large granular casts and epithelium: in the former there is but slight or no dropsical accumulation; in the latter the dropsy is extensive.

Prognosis.—Although rather unfavorable, the prognosis is not hopeless. Cases have recovered in which there had been very pronounced dropsy, and in which albumin had remained in the urine for months after the disappearance of the effusion. The more acute the symptoms and sudden the accumulation of fluid, the more favorable, provided the kidneys exhibit any activity. The prognosis is the more favorable, the shorter the duration of the disease, the less the urine departs from the standard of health, and the smaller the percentage of albumin. When the probable cause is remediable, as syphilis, or marsh-miasm, or lead-cachexia, the prognosis is favorable in proportion to the degree in which the morbid changes are due to the action of these causes.

Treatment.—A dry, unchangeable, and warm climate exercises a most favorable influence on the course and termination of chronic parenchymatous nephritis, and is a remedial agent of the first importance. When a suitable climate can not be obtained, the conditions which render it so useful should be applied to the patient, if practicable. He should be confined to bed, and remain between blankets, to secure warmth and uniformity. Free diaphoresis should be produced by warm air and by the administration of pilocarpus. If the accumulation of fluid is excessive, free purgation will be necessary, but this measure can not be continued for any lengthened period, since the implication of the mucous membrane is such that, without purgatives, there occurs a highly irritable state of the intestinal canal. Besides diaphoresis, the only resource now remaining is, to stimulate diuresis. The choice of diuretics is restricted to those which do not increase the blood-pressure in the kidneys—as the free imbibition of fluids, milk, bitartrate-of-potassa solution, etc. The infusion of digitalis, notwithstanding the theoretical objections to it, is often very serviceable in exciting free diuresis. Combination with the bitartrate or acetate of potassa increases the action of both agents. If there be great distention of the cavities and increasing difficulty of breathing, the aspirator may be used freely to draw off sufficient fluid to afford relief, but it is not desirable to empty the cavities. The removal of the fluid in the peritoneal cavity usually suffices, since the upward pressure of the ascites is the chief factor in the difficulty of breathing. Puncture of the skin may be necessary when the penis and scrotum are greatly distended, but care must be used lest sloughing follow. A small sewing-needle is employed to puncture the skin, but Southey's trocar may be used, as it is a neat, elegant, and efficient instrument for the purpose. If the fluid can be removed by the application of these remedies, iron should now be used to correct the anæmia. Combination with iron increases the action of diuretics. As the presence of albumin after the disappearance of the dropsy indicates the persistence of the mischief in the kidneys, it is then necessary to employ remedies to check the waste of material and to remove the cause on which it depends. This is a difficult if not an impossible task. The author has had promising results from the careful administration of tincture of cantharides—five drops *ter in die*, and continued, if the results are favorable, for several months ; still more valuable has proved the chloride of gold and sodium, with or without a minute quantity of corrosive sublimate—$\frac{1}{15}$ grain of the former and $\frac{1}{30}$ grain of the latter *ter in die*. Recent reports have favored the use of methaniline, but the author's experience has not been confirmatory. Good results have also been claimed for the *Blatta Orientalis*—the cockroach—a new remedy which comes to us from Russia.

INTERSTITIAL NEPHRITIS—SCLEROSIS OF THE KIDNEYS.

Definition.—Interstitial nephritis is one of the chronic forms of Bright's disease. Various designations have been applied to it : fibroid kidney, renal cirrhosis, contracting kidney, granular kidneys, etc. The terms above given—*interstitial nephritis* and *sclerosis of the kidneys*—are correct, since they designate the seat and character of the morbid change—an inflammation of the connective tissue of the kidney, the subsequent atrophy being due to the contraction and pressure of the new elements.

Etiology—This disease, like its congener, sclerosis of the liver, is a malady of middle life, according to Dickinson occurring with greatest frequency at fifty, and rarely before twenty. As regards sex, this disease is twice as frequent in men as in women (Dickinson *), and, according to German writers, four times more frequent in men (Bartels). Social condition does not appear to have any relation to its production, as it occurs under all circumstances in life. Gout seems to have an important position as a cause ; in sixty-nine fatal cases there were sixteen due to or accompanied by gout (Dickinson). The gouty condition is produced in a considerable proportion of those exposed to emanations from lead, and gouty kidney or granular kidney occurs in an astonishingly large number of such subjects. Out of forty-two workers in lead, dying from various causes in St. George's Hospital, twenty-six had granular kidneys (Dickinson). Lead-poisoning ranks first as a cause of this disease. It is in a high degree probable that chronic poisoning by other metals may exert a similar if not so predominant an influence in the production of this disease. Drs. Da Costa and Longstreth, † in a paper on "The State of the Ganglionic Centers in Bright's Disease," demonstrate the existence of degenerative changes in the renal ganglia. The ganglia undergo fatty degeneration and atrophy, the connective-tissue hyperplasia and the new elements pass through the same process. These lesions appear to the authors of the paper to stand in a causal relation to the renal affection. These observations have been confirmed by Dr. Saundby ‡, except that he regards the change as one of pigmentary degeneration.

The author has maintained for many years that interstitial nephritis frequently follows gonorrhœa in consequence of the injurious action on the kidneys of the oils and balsams used in its treatment. Liebermeister and Bartels have lately suggested that this relation between gonorrhœa and nephritis exists, but they suppose a transference of the catarrhal process from the bladder to the kidneys.

Pathological Anatomy.—When the disease is far advanced, the kidneys, usually both, are very much reduced in size, from six or five ounces to three or two. From this extreme to a size equal to or a little greater than the normal, the gradations are numerous. Usually both kidneys are equally affected, but it sometimes happens that the disease is more advanced in one. The capsule is thickened, opaque, and somewhat adherent. The surface of the kidney presents a granular aspect, due to the formation of a great number of spherical prominences, one tenth of an inch in size generally, but they may be either larger or smaller than this figure. These prominences are grayish in color and without vascularity, but the depressions between them are very vascular. Cysts of various sizes and in varying numbers are seen here and there on the surface ; they are clear, transparent, and of a straw-color. On section, the tissue of the kidney is found to be tough and resistant. The cortical portion is thin by reason of atrophy, a line or two in thickness only remaining. The color is dark-brownish, or reddish-brown, or a yellowish-gray or fawn color, the variations being due chiefly to the amount of blood present in the organ. On microscopic examination, the connective tissue about the Malpighian bodies and the blood-vessels and beneath the capsule is thickened, and the tubes are compressed into mere threads. Here and there may be a tube complete, its epithelium intact, but large spaces exist between, consisting exclusively of fibrous tissue, with the mere remains of wasted tubes. The glomeruli are grouped in bunches owing to the wasting of the intermediate tubes, and lie imbedded in the fibrillated connective tissue. Cut off from the tubular connections, in some of them fluid accumulates, forming cysts. Interior cysts as well as those on the exterior are, however, chiefly developed from obstructed tubules.

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* "The Pathology and Treatment of Albuminuria," p. 124.

† "The American Journal of the Medical Sciences," July, 1880.

‡ "The British Medical Journal," January, 1883.

The changes are not always general, but may take place in parts of the organ; one extremity may be small, contracted, granular, the other presenting its normal appearance; the hilus may be the seat of the change and the rest of the organ be affected in patches. These examples of irregularity in the development of the sclerosis are further irregular in the fact that the kidneys are unequally involved in the morbid process. The pathological alterations are not limited to the kidneys. The left side of the heart is hypertrophied, and this succeeds to or is associated with hypertrophy of the muscular fiber of the arterioles throughout the body. The retina undergoes a form of inflammation resulting in atrophy of the optic disks, known as *retinitis albuminurica.* The changes in the vessels are an influential factor in the production of the cerebral hæmorrhage with which this disease not unfrequently terminates.

Symptoms.—The development of this disease is so slow and from such small beginnings that it is usually far advanced before any symptoms arise indicating the nature of the malady. There may be, indeed, no symptom referable to the kidneys. A patient dies from a cerebral hæmorrhage, and after death granular and contracted kidneys are found. Another has convulsive seizures, partial or general; the urine is then examined, and albumin is found in it. Another has headaches, his nose bleeds, and he suffers from indigestion, acidity, and flatulence, to which his other troubles are referred. Another passes water more frequently than seems natural, gets out of bed frequently at night, and seeks relief for these symptoms. Another suffers from attacks of difficult breathing—asthmatic they seem—or he gets out of breath on ascending the stairs or making any considerable exertion; he has also attacks of palpitation and a stridulous cough, and finds that he must elevate his head and chest to lie with any comfort at night. And still another has vertigo, headache, and disorders of vision, which come on without apparent cause. The solution of the problem is at once afforded by an examination of the urine and the discovery of albumin. Of all these initial symptoms, frequent micturition, especially at night, is the most usual. The urine in typical cases is pale, of low specific gravity, and is large in quantity. The color is faintly yellow, or it is colorless, of very feeble acid reaction or neutral, and the specific gravity falls to 1003 to 1010. While the daily quantity passed by a healthy adult is about forty ounces, in this disease the urinary discharges amount in twenty-four hours to a gallon or more. It is an ill-omen when the urinary discharge falls off considerably, for this indicates still greater damage to the kidneys, and bodes the onset of uræmia. The urine, as a rule, contains more or less albumin, but it may be absent for days together, and indeed may be absent for much of the time throughout the disease. Hence frequent examinations must be made, and at longer intervals, in doubtful cases. The amount of albumin discharged is not large at any time, and in the beginning of the morbid change in the kidney may be very small, so as to produce but a faint cloudiness, and requiring the utmost nicety of observation to detect it. The quantity of albumin is affected by diet, mode of life, and by the amount of the urinary discharge. The solid constituents of the urine, especially the urea, are much reduced; uric acid is also present in very small quantity, the saline constituents are equally light, and the phosphoric acid is especially very much below the normal. Hence the urine appears clear, like water, and deposits little sediment. There may be seen some octahedral crystals of oxalate of lime, an occasional epithelial cell, and hyaline casts. The last-mentioned constituent in the sediment is most important. The casts are few in number, and hence the sediment should be collected from a considerable quantity of urine. They are pale transparent, their outlines not easily discerned, and without structure, except an occasional adherent granule or fat-globule. These pale hyaline casts must be distinguished from the pale yellow and highly refracting casts which appear in the urine in parenchymatous nephritis.

Sufficient facts have now been accumulated to render it certain that there is a form of chronic interstitial nephritis without the presence of albumin in the urine. All the other symptoms are, however, quite distinct: the copious flow of pale, watery urine of low specific gravity, the high tension of the vessels, the hypertrophied arterioles, the enlarged heart, the disorders of vision, retinal changes, the headache and other nervous phenomena, the neuroses of the respiratory organs, the disorders of digestion, the failure of nutrition, and the general decline in health and strength—indeed, all the characteristic symptoms, except the presence of albumin, are encountered in full severity.

At first, in this disease, the appetite and digestion are good, and the nutrition of the body continues unimpaired. Thirst is an early symptom. More fluid is taken at meals, and at other times a quantity of water, which seems to the patient to pass through the body without a halt. Presently, distress after eating, even epigastric pain, flatulence, and irregularity in the stools, are experienced. Acidity, pyrosis, depressing nausea, with headache, come, as the case progresses, to be very constant symptoms. The body-weight declines, the skin becomes dry, scurfy, and of a dead yellowish-white or fawn color, and the hair appears dry and lifeless. The strength fails, and the breathing becomes labored on making any exertion. This is due partly to the losses of material and partly to the changes occurring in the heart. The left cavities undergo hypertrophy, and the arterioles throughout the body are in a state of abnormally high tension, owing to hypertrophy of their muscular layer ; hence the radial pulse exhibits an exalted tension and force. Much discussion has occurred as to the existence of this thickening of the muscular fibers of the tunica media, and as to the causes, but the fact seems now firmly established. The obstacle to the circulation produced by the abnormal tension in the arterioles is the chief if not the only factor in causing hypertrophy of the left ventricle. Toward the end, however, a change takes place in the hypertrophied muscle : it undergoes fatty degeneration ; then the cardiac movements become weak, the sounds indistinct, and the circulation feeble. In this form of kidney-disease there is usually no dropsy. It is true, œdema may occur from various complicating conditions, if not from the kidney-disease. When urine can no longer be separated from the blood by the damaged organs there will be dropsy, but death takes place with the phenomena of uræmia. When some lesion of a valve occurs, especially if of the mitral, œdema will appear in the ankles and face. Pleural inflammation or hepatic disease may result respectively in hydrothorax or ascites. Although the dropsy is never sufficient to cause death—is never anything more than an œdema of the face and extremities—yet death may be due to a sudden œdema of the lungs. When the case is approaching its termination, the symptoms of uræmia develop. The nausea which had existed before, with occasional vomiting, increases, becomes incessant, and the vomiting is violent and uncontrollable. The vomiting is not necessarily excited by the presence of food ; it occurs when the stomach is empty, in the early morning ; and after severe and protracted retching only a little mucus, with a quantity of watery fluid of low specific gravity and very feeble acidity, comes up. Diarrhœa also now gradually increases, and toward the end becomes uncontrollable, the stools being thin, abundant, and frequent. At last the evacuations consist of a watery fluid, with some mucus, and very little fecal matter, and occur involuntarily. The vomiting and purging are largely vicarious of the urinary secretion, which contains less and less solid matter. The profuse discharges are very exhausting, and consequently serve to develop the symptoms proper to uræmia. There is, now, an increasing headache ; much vertigo is experienced ; hebetude of mind and a soporose state came on, so that when his attention is withdrawn from persons and things the patient falls asleep in his chair, but sleep at night is disturbed by vivid dreams, and there are much muscular twitching, jerking, and heavy, irregular breathing. Unsymmetrical convulsive movements, jactitations of individual muscles and groups of muscles, of the face or extremities, and general convulsions, occur as the case approaches the end. The patient when fully aroused may still be entirely conscious, but he soon lapses into stupor when left to himself; there may be maniacal delirium and violent struggling, or unconsciousness between the convulsive seizures. An early symptom in many cases of interstitial nephritis is amblyopia, double vision, hemiopia, and other derangements of vision. As has been pointed out, these symptoms may be the first to attract attention, so that the diagnosis is made by the oculist. When the examination is made by the ophthalmoscope at an early period, the optic disks are found to be swollen; the veins are enlarged and tortuous, while the arteries are rather shrunken. Whitish spots appear on the retina, of various sizes, and hæmorrhagic extravasations occur along the vessels, but both chiefly about the disks and in the neighborhood of the macula lutea. Both eyes are affected, but in varying degrees.* While these obvious changes occur during the course of the disease, and are permanent, there are fugitive attacks in which vision may be lost without any retinal changes. Just as there may be muscular twitchings, and even convulsions, without any permament lesions, so there may be entire loss of vision without any alterations of the retina.

Course, Duration, and Termination.—Interstitial nephritis is a very chronic malady. There is a long period (often several years) from the beginning of frequent micturition to the occurrence of impaired functions elsewhere. In those cases marked, as has been pointed out, by violent initial symptoms, the disease in the kidneys has proceeded silently, and, interfering with no function, has caused no disturbance until the sudden outbreak. It sometimes happens that a man falls in the street, is violently convulsed, and dies in a few hours comatose, the real lesion in the kidney having gone on unobserved for months and years, it may be. The duration of the disease can not, therefore, be definitely expressed. The termination is most usually with uræmia—convulsions, coma, and death. The changes in the vessels and the hypertrophy of the heart are the causes of cerebral hæmorrhage with which many cases end. The excrementitious matters circulating in the blood give rise to inflammations of the serous membranes, notably pericarditis and endocarditis, which prove fatal. Death may be caused by hæmorrhages from the mucous surfaces, or from the exhaustion caused by violent vomiting and purging.

Diagnosis.—The recognition of this disease, when the existence of albuminuria has been ascertained, can never be difficult. The large quantity of urine, the absence of color, the low specific gravity, the small amount of albumin, the hyaline casts, the hypertrophied heart and arterioles, are to be compared with the small quantity of urine, the high color, the high specific gravity, the immense quantity of albumin and granular casts, the rapid, large, and general accumulation of fluid. These prominent features from the clinical standpoint readily separate interstitial and parenchymatous nephritis. Pathologically, the small, tough, granular kidney and the large, soft, pale, and smooth kidney are perfectly distinct.

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* "On the Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys," Dr. T. Clifford Allbutt, chapter vii, London: Macmillan & Co.

Treatment.—As interstitial nephritis is an incurable disorder when the proper secreting structure of the organ is destroyed, it is important to arrest the initial changes, if we possess the means of so doing. Those cases arising from syphilitic infection, or from plumbic or other metallic poisoning, offer the best prospect of cure, if the proper remedies are applied. It is in the cases arising from these causes, probably, that such good results are obtained by the persistent use of full doses of the iodide of potassium. The author has observed several cases in which the iodides seemed to arrest the disease permanently, and others in which the corrosive chloride, administered in small quantity (one twentieth of a grain) for a lengthened period, effected cures under apparently very unpromising circumstances. Better results even, the author believes, are procured from the careful and persistent administration of the chloride of gold and sodium. Very unpromising cases have, apparently, yielded to this remedy. It is usually given in pill, $\frac{1}{15}$ to $\frac{1}{20}$ grain being given three times a day persistently. Similar therapeutical properties are possessed by arsenic. In sclerosis of the liver, as well as in that of the kidney, we find that arsenic exercises a favorable influence in retarding the changes. This remedy is all the more desirable since it has, in small doses, a sedative effect on the stomach, and promotes appetite and digestion. These remedies, intended to arrest the hyperplasia of the connective tissue, should be prescribed with a definite relation to the presumed cause—iodide of potassium and bichloride of mercury, in those with a syphilitic history; iodide of potassium, in those poisoned by lead; and chloride of gold and arsenic, in those cases of unknown origin. When there are much acidity, flatulence, and pain after food, mineral acids, especially the nitric, taken before meals render important service. Doubtless the uric-acid diathesis is a very influential factor in the development of the disease, and hence those remedies which lessen its formation are deserving of high consideration. The utility of the mineral acids consists in preventing the acid fermentation of the food and in promoting digestion, so that the nitrogenous constituents are better prepared for assimilation. For the anæmia present, iron is generally prescribed, but the effects are usually rather disappointing. The most useful chalybeate is the tincture ferri acetata, which is also formed extemporaneously in Basham's mixture, composed of tinct. ferri chloridi, liquor ammoniæ acetatis, and acetic acid. If iron is given freely and for a long time, headache and a disordered stomach will require its discontinuance; nevertheless, the occasional and careful use of iron is beneficial. Remarkable results have been achieved by the use of nitro-glycerine. It lessens the high tension of the vessels, relieves the pain in the head, and removes many of the disagreeable subjective sensations. By dilating the arterioles this remedy probably, also, improves the nutrition of the kidneys. The author has usually given this remedy in the form of the one per cent. solution, beginning with one drop and increasing the dose until its physiological effects are experienced. The dose in this form of the remedy is more easily regulated. The chloride of gold and sodium may be administered coincidently. When the symptoms of uræmia come on, the case requires most careful handling. If the stomach and intestines are yet capable of good work, the treatment may be more direct and efficient; but if the severe, even uncontrollable vomiting and purging occur, so often present as a part of the uræmia, the difficulties of the management are greatly enhanced. In the former case, active purgatives, as elaterium, croton-oil, and compound jalap powder, procure elimination through the intestinal canal, and are of signal service. In the latter case, the important results derived from purgatives are precluded. Diaphoretics, as the vapor or hot-air bath and the injection subcutaneously of pilocarpine, are the most powerful means of relief. Purgatives and the vapor-bath, or pilocarpine, will in those cases of acute exacerbation in the renal trouble, when the patient is yet in good condition, relieve the symptoms remarkably, and subsequently there may be a long period of tolerable health. The convulsive and nervous phenomena of uræmia are best remedied by the means for procuring elimination, but, if the symptoms are urgent, the inhalation of amyl nitrite, chloroform, and ether may be necessary. The hypodermatic injection of morphine in large doses has been shown by Loomis, of New York, to have a remarkable influence on the convulsions of uræmia; but chloral by the stomach or rectum may be better.

The nutrition of the patient is of the first consequence. The diet should be simple, and consist of milk, eggs, a little fresh meat (once a day), and fruits, if diarrhœa does not exist. The best results have been obtained from an exclusive milk-diet; as this becomes irksome, intolerable even, the plan of diet just suggested is best. Malt liquors, spirits, and wines are highly objectionable, especially the first named. The clothing should be warm; flannel should be worn by day, and the patient should sleep between blankets. Whenever his means will permit, the patient should seek a warm, dry, and uniform climate. Recent observations by Drs. Sparks and Bruce in respect to the influence of diet, rest, and exercise, on the excretion of albumin, have led to the following results: the amount of albumin is much reduced by a milk-diet and non-nitrogenous food, and "absolute rest remarkably reduced the amount of albumin."*

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* "Medico-Chirurgical Transactions," 1879, p. 254.

HÆMATURIA—HÆMATINURIA—HÆMOGLOBINURIA.

Definition.—By *hæmaturia* is meant the discharge of blood in the urine. It is a symptom rather than a disease, and is discussed in connection with maladies of which it is a part. *Hæmatinuria* signifies the presence of blood coloring matter in the urine, and is a paroxysmal affection, accompanied by constitutional disturbance, and most frequently caused by malarial toxæmia. It is also designated *hæmoglobinuria*.

Pathogeny.—Sex is an important factor in the pathogeny of this affection, males being most freqently (ten to one) the subjects of it. Youth and adult life are the periods during which it appears, and after fifty it is very uncommon. The chief cause of hæmatinuria is malarial poisoning, and hence the disease is encountered within the malarial zone. In the Southern States, especially in Alabama, it is becoming more frequent, and the severe or malignant form is now comparatively common. As a rule, it is irregularly intermittent, but there are many exceptions to the rule in that the morbid manifestations are remittent or continuous.

Although regarded generally as a modification of the blood, there are recent observations which tend to prove that a form of nephritis is also necessary.* The essential condition is a disorganization of the blood-corpuscles, separation of the hæmatin, and such a change in the walls of the capillaries as to permit transudation of these elements.

Symptoms.—The attacks of hæmatinuria are severe in proportion to the local activity of the malarial poison. Within the tropics the maximum violence of all the symptoms is reached. It is usual to describe two forms—the mild and the severe—but it is quite certain that between the extremes there are numerous gradations in the severity of the manifestations dependent on the climatic conditions. The persons attacked, boys or men usually, have been exposed to malarial infection if not attacked with malarial fever. In the mildest cases the onset of the hæmatinuria is announced by the sudden appearance of bloody-looking urine, some lassitude, chilliness or coldness of the hands and feet, muscular pains and weariness, yawning, blue lips and finger-nails, followed by a reaction stage, fever, terminating in a sweat of greater or less severity. With the subsidence of these symptoms, the urine changes in appearance, the blood coloring matter disappears and the normal is restored, and the general health may be entirely recovered. In the severest form the chill approaches the congestive in character; there is profound depression of the powers of life; vomiting occurs; the urine becomes deeply colored, the skin yellow, and the fever high, remittent in type, or passing into continued.

The most characteristic feature in every way, and that which gives it most importance, is the appearance of the urine. The color varies from a slight reddish tinge to a deep port-wine, and the deposit which collects on standing is composed of the *débris* of disintegrated blood-corpuscles, epithelium of the tubules, amorphous urates, and sometimes brownish casts of a hyaline character, or made up of disintegrating blood-globules, or pigment-matter. Albumin is present in considerable quantity, and blood-disks sometimes, but usually the bloody appearance of the urine is due to the blood-pigment in a granular form or dissolved, and the reaction is decidedly acid, rarely alkaline. When the paroxysm is over, the abnormal coloration lessens, then ceases, the albumin disappears, and in a few hours the urine becomes healthy. When the next paroxysm comes on the same phenomena occur. The malarial poison must then, directly, or through the agency of some substance formed in the intestinal canal, act on the blood-corpuscles, extract the hæmoglobin which is dissolved in the serum and eliminated by the kidneys, or which destroys the corpuscles in the liver, spleen, and kidneys. When the blood is examined, the white blood-disks are found to be increased in number, and the red are paler in tint, and exhibit no tendency to form the characteristic *rouleaux*, or the color disappears entirely, and the disks are changed in shape.

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* Ponfick, Virchow's "Archiv," vol. lxxxviii, p. 476. Also, Lebedeff. *Ibid.*, vol. xci, p. 267.

Urticaria often manifests itself, especially on parts exposed to cold, and the skin has either an icterode hue or is deeply jaundiced, which passes off after the systemic disturbance is entirely ended.

Course, Duration and Termination.—As seen in this country, hæmatinuria is a distinctly periodical affection; occurring with considerable regularity, and accompanied by the usual systemic disturbances of a malarial fever. Not all the cases are regularly intermittent and paroxysmal; some of them are remittent; a few continued in type. The intermittent cases may be free from fever, the urinary symptoms apparently taking the place of the usual chill, fever, and sweat. It has no fixed period of duration, and may last indefinitely, but much depends on the rôle of malaria in its production, and the appropriate use of quinine. In the author's experience it has a decided tendency to recur again and again, after it has been entirely relieved, and the normal condition of the urine and the blood restored. Chronic nephritis is an outcome of some cases, and intercurrent diseases, especially pneumonia, are apt to occur.

Diagnosis.—Hæmatinuria is distinguished from hæmaturia, with which, necessarily, it is most frequently confounded, by the presence of the blood-pigment without the blood-globules, at least in their normal form, by its periodical character, by the absence of the local lesions producing the latter, and by the presence of casts in the urine. Cancer of the kidney is accompanied by hæmaturia, but in that case blood is present, the discharge of blood is not periodical, and fever is wanting. Cancer is accompanied by a marked cachexia, by decline in health and strength before the local symptoms assume importance; hæmatinuria as a rule does not impair the vital forces to any considerable extent, except during the paroxysms, and after these are ended a prompt return to the normal occurs.

Treatment.—The most important remedy is quinine, and that it must be given in large doses is the conclusion of the physicians of the South, who have the largest experience,[*] and is the author's judgment, after considerable observation of the disease. The patient must be cinchonized, in anticipation of the seizure, whenever the time for recurrence can be fixed on, and attention must be given to the "septenary periods," so called, that the outbreaks which may occur at certain times, following the law of evolution of the malarial poison, may be prevented. Dr. Narcom advocates the use of morphine, and his view is confirmed by others. It may be given with the quinine, or administered hypodermatically at the onset of a paroxysm. According to Ralfe,[†] arsenic is a remedy of great value, not so much for the immediate relief of the paroxysms as for the improvement of the patient's condition and the prevention of subsequent seizures. He administers it persistently in the intervals. A combination of quinine, arsenic, and ergot, given regularly, for several months if need be, has a remarkable effect in bringing about improvement and cure.[‡] Such a combination should be administered after the impression made by large doses of quinine has been effected.

THE AMYLOID DISEASE OF THE KIDNEYS.

Definition.—By the term *amyloid disease* is meant an affection characterized by the deposit of amyloid matter. As it occurs in the kidneys, this disease is known as *lardaceous kidney*, *waxy kidney*, because of the supposed resemblance to lard and wax respectively. By Dickinson the disease is distinguished by the title "depurative infiltration."

Causes.—The chief cause is suppuration, especially protracted suppuration of or connected with the cancellous structure of bones, or ulcerations affecting the skin and mucous membrane. It is necessary that the suppuration be profuse and protracted, but it is not necessary that it occur in bone only. But suppuration alone is not sufficient to cause the amyloid deposit. There must be a peculiarity of constitution precedent, for, of all exposed to this destructive malady by suppuration, but a small number actually are affected by amyloid change. It is more apt to occur in those under the influence of chronic malarial poisoning, but more influential diathetic states are those of syphilis, scrofula, tuberculosis, and cancer—especially cancer.* It is impossible to indicate in the present state of knowledge the relation of these cachexiæ to amyloid disease, but it seems pretty clear that more or less protracted suppuration coincided with the cachexia. According to Bartels, ulcerations of the intestines are more certain than ulcerations of any other mucous membrane to induce amyloid disease; and, further, that the suppurating center must have communication with air to possess this peculiar property. The amyloid deposits are not limited to one organ, but occur in the liver, spleen, intestinal canal, the supra-renal bodies, the lymphatic glands, the thyroid gland, and the kidneys.

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[*] Dr. Thomas J. Turpin, of Forkland, Alabama, "The Treatment of Hæmorrhagic Malarial Fever" (pamphlet); Dr. W. A. B. Narcom, "Transactions of the North Carolina Medical Association" for 1874.

[†] "A Practical Treatise on Diseases of the Kidneys." London, 1885, p. 552.

[‡] ℞ Quininæ sulph., ʒj; ext. ergotæ (Squibb), ℈j; ferri arseniat., gr. iij. M. Ft pil. no. xx. Sig.: One pill three times a day.

Pathological Anatomy.—The term *amyloid*, or starch-like, was originally proposed by Virchow, because of the reaction under iodine, and the characteristic structure remotely resembling starch. The theory of Dickinson that this substance is fibrin deprived of its alkali, which has been eliminated from the body in the pus, has been completely disproved by the elaborate investigations of Mr. George Budd.† "The cells of an organ affected may be seen to become gradually distended with a translucent deposit, and soon an accumulation of a similar deposit takes place in the intercellular spaces also." There is present in the blood in the normal a considerable quantity of a substance, named by Seegen "dystropodextrin"—"a substance which agrees with lardacein (amyloid material) in its most specific characteristic." To account for lardaceous disease, then, it is only necessary to suppose that this dystropodextrin becomes insoluble, and is precipitated and deposited in the tissues. This substance reacts to iodine, just as the amyloid matter, and agrees with it in all other particulars, so that this theory is more plausible than any heretofore proposed. When the lardacein is deposited in the kidneys to a considerable extent, the organs are larger and heavier than normal, and are also very firm in texture. The capsule, which is very thin, is easily detached, and the surface of the kidney is pale, gray, or whitish, and has a glistening, even a polished, appearance. The cortical part is broad, but pale and anæmic, while the cones are dark and congested. On microscopic examination, the change that has taken place in the organ is found to have occurred along the renal vessels and in the vascular tufts of the glomeruli, at first at isolated points, and subsequently along the whole extent of these vessels. As the morbid proces extends, the afferent and efferent vessels, the vasa recta, and ultimately the renal epithelium and even casts, still contained within the tubes, are seen to be embraced in the degeneration or deposition. If a thin section of the kidney is laid on a white plate after being brushed over with the iodine solution (iodine and iodide of potassium), the branching lines and points of reddish-brown stand out prominently beside the pale yellow of the healthy tissues.* Besides the kidneys, other organs of the body undergo the same change, but the kidneys may be affected alone. The supra-renal capsules, the liver, spleen, the intestinal canal, etc., are similarly affected. When an organ is thus infiltrated by this new material, its proper structure undergoes an atrophic degeneration by pressure. With the amyloid change may be associated interstitial or parenchymatous nephritis, especially the latter. It is more proper to say that during the progress of interstitial nephritis the amyloid degeneration comes on; hence the lardaceous or amyloid kidney may be more or less granular and contracted, instead of being enlarged and smooth. With lardaceous kidney are associated chronic ulceration of the lungs, and suppurating cavities, ulcerations of the intestines, diseases of bones and joints, syphilitic lesions of the mucous membrane, external integument, and scrofulous abscesses.

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* "Transactions of the Pathological Society" of London, vol. xxx, p. 511; paper by Dr. Dickinson, and discussion.

† London "Lancet," February 28 and March 27, 1880; "Amyloid Degeneration," by George Budd, Jr.

Symptoms.—As amyloid disease of the kidney arises during the course of some chronic wasting malady, its onset is necessarily obscured by the complexus of symptoms already prominent. There is, of course, a marked degree of anæmia produced by prolonged suppuration, and by amyloid changes in other organs besides the kidney. The urine is, as a rule, increased in amount and may be considerably so, especially in those cases complicated by interstitial nephritis, or it may be considerably diminished in quantity, when there coexists parenchymatous nephritis. But in genuine amyloid kidney the urine is increased, is pale, watery, and of very low specific gravity—1002 not unfrequently—and usually under 1006. When associated with parenchymatous nephritis the specific gravity may rise to 1030, or when, as may happen, the quantity passed is very low. The amount of urea and other solid constituents is much reduced when the quantity is great, and greater when the quantity of urine is small. The amount of urea excreted depends on two factors: on the functional activity of the liver and the extent of disease in the kidneys. Albumin is always present. At times, during the first implication of the kidneys in the morbid process, there may be none, and when present the quantity is sufficient to impart a faint cloudiness merely, but it becomes permanent as a constituent of the urine during the height of the disease, unless just at the close, when it may disappear again. The urine contains so little else than water that the sediment is very small in amount, and hence it requires a good deal of urine to collect even a few casts. Only the hyaline casts are proper to this disease; they are perfectly transparent, homogeneous, and slender, so that they are seen only by careful management of the light. Large granular casts, blood-corpuscles, and renal epithelium may be present in considerable quantity when parenchymatous nephritis is a complication. The casts may present a faintly yellow and highly refracting appearance when attacked by the amyloid change or composed of the amyloid material.

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* Safranine, an aniline product, is said to be an admirable test for amyloid matter. Sections are immersed in a very dilute watery solution. The amyloid matter is stained orange-yellow; the rest of the tissue, rose.

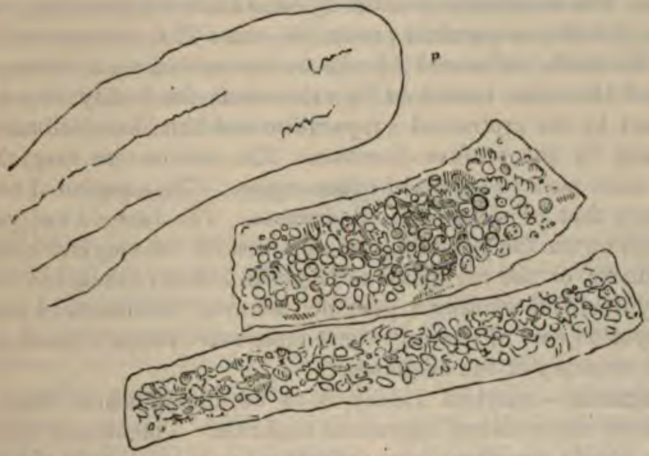

FIG. 41.—A Large Hyaline Cast without, and Two with Epithelium. (Beale.)

More or less œdema is always present, but general dropsy is infrequent. The œdema is found in the lower extremities, and ascites is usually present, and disproportionate to the quantity of fluid elsewhere. This is doubtless due to the implication of the liver in the general morbid process, and to the swelling of the lymphatics in the hilus of the liver, compressing the vena porta. With the progress of the disease, there are necessarily increasing weakness and anæmia, a peculiar earthy or fawn color of the skin, and pigmentation of the eyelids. The exhaustion of the vital forces is greatly hastened by the occurrence of a profuse, watery, and uncontrollable diarrhœa. Vomiting also occasionally takes place, but not nearly with the frequency and persistence of the diarrhœa.

Course, Duration, and Termination.—Amyloid kidney is an essentially chronic malady, but its fortunes partake of the changes and progress of the associated malady. Commencing insidiously, its presence is recognized only when an increasing urinary discharge calls attention to the state of the kidneys. The duration of the disease is largely determined by the suppurating malady causing it; but, when the amyloid change is clearly begun, the case usually terminates in death in a few months, but may extend to years. Uræmia, as manifest in vomiting, purging, amaurosis, partial and general convulsions, etc., does not occur in amyloid disease, unless the contracting kidney also develops, or there is a sudden appearance of parenchymatous nephritis. Death by cerebral hæmorrhage is also rare. Hypertrophy of the heart and of the arterioles does not take place in this form of kidney-disease. The termination is often by some acute inflammation, as pneumonia, pleuritis, or purulent peritonitis, etc. The duration will necessarily be much influenced by the occurrence of such inflammation. Many of the cases terminate by exhaustion, the bodily forces being worn out by the protracted suppuration and the loss of albumin, but especially by the profuse diarrhœa. The termination may, then, be due in most cases to lesions of other organs. The question of recovery is largely that of the associated diseases. The data do not yet exist for deciding on the possibility of an arrest of the amyloid change in the kidneys, or the regression of deposits already made, but it is extremely doubtful whether a genuine case ever terminates in recovery. In a reported case of recovery there must ever remain a doubt respecting the accuracy of the diagnosis.

Diagnosis.—Amyloid kidney is to be distinguished from parenchymatous nephritis and interstitial nephritis. The history of the case is here highly important, especially the constant relation of suppuration to lardaceous degeneration. In parenchymatous nephritis the urine is scanty, high-colored, of high specific gravity, and deposits an abundant sediment, containing urates, granular casts, tubular epithelium, and red-blood globules; in amyloid kidney the urine is abundant, pale, of low specific gravity, deposits very little sediment, containing a few hyaline casts and occasional waxy casts, but no blood-corpuscles. In parenchymatous nephritis, dropsy forms quickly and is extensive; in amyloid kidney, the effusion is slight and confined to the lower extremities and to the peritoneal cavity. Amyloid kidney is distinguished from chronic interstitial nephritis by its history and association with suppuration in some form, and with the evidences of the same change in the liver, spleen, and intestinal canal. In chronic interstitial nephritis the symptoms of uræmia are very pronounced at some period; in amyloid kidney these symptoms very rarely occur at any period.

Treatment.—As when the amyloid deposits have taken place it seems doubtful if their removal can be effected, it is highly important to stop all sources of suppuration, and thus prevent the deposition of the altered fibrin. Attention should be directed at once to the cure of suppuration. As syphilis and the suppuration connected with it are a fruitful source of mischief in this direction, this malady should be efficiently treated and cured, and all cases presenting a syphilitic history should be given a thorough course of the iodide of potassium.

Dickinson, influenced by his theoretical notions, advises the internal use of the potash and soda salts, supplying artificially the alkali which is carried off in the pus, while the fibrin is deprived of it. He at the same time enjoins the free use of eggs and milk, to supply the material lost in the urine. A combination of the chloride of gold and sodium ($\frac{1}{16}$ gr.) and bichloride of mercury ($\frac{1}{30}$ gr.), persistently administered, accomplishes more than any other remedies. Iron, cod-liver oil, and a generous diet are demanded by the condition of feebleness and anæmia. The exhausting diarrhœa resists all means of treatment, but the most efficient remedy, according to the author's experience, is Fowler's solution and opium tincture—three drops of the former and five to ten of the latter, three or four times a day.

PYELITIS AND PYELONEPHRITIS.

Definition.—*Pyelitis* means an inflammation of the pelvis of the kidney; *pyelonephritis* includes pyelitis and a consecutive or simultaneous suppurative inflammation of the kidneys. They are here considered together to avoid repetition, and because of their frequent association.

Causes.—Probably the most frequent cause of pyelitis is the extension of a morbid process from the bladder to the pelvis of the kidney, by the ureter. Catarrh of the bladder is lighted up by decomposition of the urine, consequent on its retention. Whenever an obstacle exists to the discharge of urine from the bladder, the decomposition ensues, the urine becomes ammoniacal, and the mucous membrane the seat of an active catarrhal process. Stricture of the urethra, enlarged prostate, the pressure of the retroverted uterus, pregnant uterus, or of a pelvic tumor, etc., act by hindering the urinary discharge. An inflammation of the mucous membrane of the bladder, due to gonorrhœa or other causes, will have the same effect by causing fermentation of the urine. A renal calculus, or other foreign body, present in the pelvis of the kidney, will produce catarrh directly by irritating the mucous membrane. Decomposition of the urine and catarrh extending to the pelvis of the kidney are produced by paraplegia: the bladder being paralyzed, the urine is retained and undergoes putrefactive fermentation. Diuretics of the stimulant kind, as copaiba, turpentine, and cantharides, irritate the mucous membrane of the pelvis of the kidney in passing through these organs. Whenever the urine decomposes, vibrios and bacteria appear in it in immense numbers; the urea is decomposed and converted into the carbonate of ammonia; the ammoniaco-magnesian phosphate crystals are formed in great quantity, and much phosphate of lime is separated by the inflamed mucous membrane. Pyelitis occurs as a complication in various infective maladies—in pyæmia, puerperal fever, the exanthemata, etc., and may result from the extension of a neighboring inflammation.

Pathological Anatomy.—The changes consist in the ordinary catarrhal process, the mucosa and the submucosa becoming very much thickened in old cases, the vessels varicose, and the epithelium much changed by the proliferation of its cells, etc. If the morbid process began in the bladder, the evidence will be plain, and the ureters may or may not be affected by the same changes. If the pyelitis has existed for some time, the kidneys will be seen to be in a process of suppuration—one or both. The organ is more or less enlarged, is deeply congested and reddish, except certain spots which present a yellowish-white color, are wedge-shaped, and extend through the cortex to the apex of the cone. On section these patches present here and there points of suppuration, are swollen, and the capsule is more or less firmly adherent to them. Suppuration occurs soon all along the extent of these patches between the tubules. Several of these suppurating patches uniting, considerable abscesses form ; the kidney elements are disassociated, broken up, and disappear ; and from the cones the suppuration proceeding destroys the cortical part, and ultimately nothing remains but a bag of pus having irregular walls marked by septa, remains of calyces. It seems well established that the suppurative inflammation in the kidneys is set up by the presence of bacterian colonies which have migrated from the inflamed bladder. With high powers the bacteria are seen arranged in parallel lines within the tubules. They appear as minute, globular, highly refracting granules. After a time the same bodies are seen in the interstices with pus-corpuscles. The epithelium of the tubules is at first cloudy, granular from fatty degeneration, but is soon destroyed, the whole tube being filled with the branching filaments and spores. According to Klebs (Ebstein), the inflammation proceeding to suppuration is excited by the bacteria.

Symptoms.—The pyelitis or pyelonephritis usually encountered is associated with chronic cystitis, ammoniacal urine, and the systemic state produced thereby. When due to the presence of a calculus in the pelvis of a kidney, the symptoms are different in some respects ; hence the consideration of this form is properly postponed to the section devoted to this topic. In the form of pyelitis now under consideration, there is usually more or less irritability of the bladder, and the urine is somewhat more abundant than normal. The urine is neutral or alkaline in reaction, milky in appearance when voided, and deposits a copious sediment, whitish or faintly yellowish-white in color. The upper layer of the sediment is more distinctly whitish, lighter, and easily disturbed with a little agitation, whereas the bottom layer is heavier, firmer, and unites in an homogeneous mass which sticks closely to the vessel, and when dislodged rolls out in a tenacious, gelatinous mass. There is some albumin present, but not more than is proper to pus. On microscopic examination there are present mucus and pus-corpuscles, chiefly large crystals of ammoniaco-magnesian phosphate, and by no means frequently epithelial cells from the pelvis of the kidneys. In the form of pyelitis arising from decomposing urine in the bladder, it is difficult to find the morphotic elements belonging to the kidney. Besides the corpuscular and crystalline forms above mentioned, the urine contains numberless bacteria. There is more or less uneasiness felt posteriorly just under the false ribs and extending downward along the course of the ureters, and the usual distress arising from the bladder under these circumstances. The strength declines, the body loses flesh, and there is more or less fever, increasing toward evening and with a morning remission. In some cases, when pyelonephritis is developed and suppuration is going on in the kidney, the fever has a distinct typhoid type, and has been mistaken for typhoid; for the cerebral disturbance—low-muttering delirium—*subsultus tendinum*, and stupor, due to uræmia, come on with septicæmic fever, diarrhœa, and exhaustion, due to suppuration. In still a third group the symptoms are those of pyæmia. Chills occur at irregular intervals, followed by very high temperature, the thermometer indicating 104°, 105°, or 106° Fahr., and then a profuse sweat. The face has an earthy hue, the countenance is anxious, and the features are retracted and pinched. The exhaustion is extreme, the pulse feeble and rapid. During the febrile exacerbation there is usually more or less delirium. A profuse diarrhœa and complete anorexia hasten the decline. Secondary abscesses may form in the articulations, or in the intermuscular septa, which increase the already rapid tendency downward.

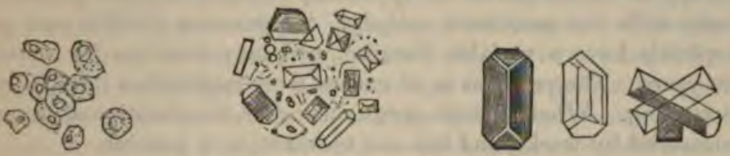

FIG. 42.—Various Forms seen in Pyelitis.

Besides the usual form of pyelitis and pyelonephritis associated with the various obstacles to the outflow of urine, and with ammoniacal and decomposing urine, there are several milder forms. Certain renal irritants, as cantharides, turpentine, etc., and exposure of the body to cold while in a warm and perspiring state, will produce a simple, primary, acute pyelitis. There occurs more or less pain in the region of the kidneys, extending downward along the course of the ureters, and there may be slight feverishness toward evening. The urine is acid and somewhat increased in quantity. It deposits a sediment composed of urates, pus, and occasional blood-corpuscles, and epithelium from the pelvis of the kidney. Pyelitis also occurs in childbed. Then it begins with chill, followed by fever, and pain in the lumbar region. The pain may have a very acute character, and, shooting down along the ureters into the bladder, seem like nephritic colic. The urine is little changed from normal, but it contains some pus and cells of renal epithelium.

Course, Duration, and Termination.—The simple cases of pyelitis terminate in recovery in from one to two weeks. Those occurring in childbed, or in the course of typhoid, puerperal, or other fevers, terminate with the associated malady. Suppurative pyelitis and pyelonephritis have a variable duration, and may continue for months, even years. The progress is, of course, more rapid when the kidney is suppurating. When uræmic symptoms occur, the duration of the case is measured by weeks, and but one termination is possible.

Diagnosis.—In the most common variety the diagnosis is often merely conjectural, for the muco-pus is so abundant that it is extremely difficult to find the characteristic forms from the pelvis. When uræmic symptoms finally come on, there can be doubt no longer. In the simple cases the diagnosis must rest on the association of pain, with altered urinary secretion, the epithelium of the pelvis of the kidney being present.

Treatment.—In the simple cases mere dilution of the urine affords relief. If the urine is acid, a potash salt—liq. potassii citratis—should be administered freely. In the cases of pyelitis associated with ammoniacal urine, benzoic acid is extremely serviceable. Gallic acid, passing through the kidneys unchanged, has a local effect of a very useful kind. Excellent results have been obtained from the persistent use of eucalyptol, or fluid extract of eucalyptus. The oils of turpentine, copaiba, and cubeb have a good effect in changing the character of the mucous membrane and limiting the formation of pus; but they must be given in small doses. Quinine has a high degree of utility—to keep down the abnormal temperature, to support the powers of life, and to check pus-forming. It is important throughout to keep up the strength by suitable aliment.

RENAL CALCULI—NEPHROLITHIASIS.

Definition.—*Renal calculi* are concretions formed by precipitation of certain substances from the urine about some body or material acting as a nucleus.

Causes.—Calculi occur at all ages, and are very frequent in children before the fifth year, and from five to fifteen. Males are much more liable to them than females. A sedentary life and indulgence in a highly nitrogenized diet are circumstances favoring the occurrence of the uric-acid diathesis. Certain districts of country seem peculiarly disposing, the character of the drinking-water being held responsible, especially the lime present, but this explanation of the fact is wholly untenable. A special susceptibility exists in certain families,* various members of which may be attacked, while other families living under the same conditions are unaffected.

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* London "Lancet," December 5, 1874.

Pathogeny.—The researches of Dr. H. Vandyke Carter, Ord,* Beale, and others have demonstrated the importance of mucus in determining the precipitation of the calculous ingredients of the urine. Calculi are of all sizes—from microscopic bodies up to a concretion filling the pelvis of the kidney. Beale † has shown the importance of microscopic calculi present in the urine, as indicating similar bodies of larger size in the pelvis. In the kidneys there may be an infinitude of calculi—from mere grains of sand to concretions of considerable size. Uric-acid infarctions, triple phosphate- and carbonate-of-lime infarctions, are found in the straight tubes of the pyramids in infants, and in old men, especially those affected with the gouty diathesis. Calculi of uric acid are more frequent than any other constituent, for, although this substance exists in small quantity, it is very slightly soluble. Ord shows that uric acid, crystallizing in the presence of colloids (albumin, mucus, etc.), tends to assume a spheroidal form, and Carter that a bit of mucus is the nucleus about which the crystallization takes place. These calculi are made up of concentric layers, and may be composed wholly of uric acid, or of alternate layers of uric acid and oxalate of lime. Similar modifications are impressed on oxalate of lime, but while they tend to assume the spheriodal form in the presence of mucus they also crystallize in octohedra. The uric-acid calculi are grayish-red or reddish-brown, smooth, hard, and having a specific gravity of 1·5. The pure oxalate-of-lime calculi are very rare, are very hard in texture, rough on the exterior, of a dark-brownish color. The oxalate of lime with a nucleus of uric acid are much more common than the pure oxalate. Calculi of cystine are still more rare than those of oxalate of lime; they are comparatively soft, and have a dull-yellow or amber color. Phosphatic, next to uric, are the most frequently encountered calculi. They are very light, friable, of a dull or grayish-white, or bright white, rough, and sometimes polished. The phosphatic deposit, consisting of phosphate of lime and the ammoniaco-magnesian phosphate, often forms about a uric-acid calculus which has been present for some time. This deposition of the phosphates may be expected to take place on a uric-acid calculus which has been long present in the pelvis of the kidney, if the urine becomes alkaline. The stones may be in one, but occasionally they are found in both kidneys. In the cases which have fallen under my observation, two thirds were in the left kidney. The results of the presence of concretions differ according to their situation: in the tubules, as infarctions, they excite inflammation of the kidney; in the pelvis they cause pyelitis. Gouty kidney is a result of the uric-extends, the afferent and efferent vessels, the vasa recta, and ultimately the renal epithelium and even casts, still contained within the tubes, are seen to be embraced in the degeneration or deposition. If a thin section of the kidney is laid on a white plate after being brushed over with the iodine solution (iodine and iodide of potassium), the branching lines and points of reddish-brown stand out prominently beside the pale yellow of the healthy tissues.* Besides the kidneys, other organs of the body undergo the same change, but the kidneys may be affected alone. The supra-renal capsules, the liver, spleen, the intestinal canal, etc., are similarly affected. When an organ is thus infiltrated by this new material, its proper structure undergoes an atrophic degeneration by pressure. With the amyloid change may be associated interstitial or parenchymatous nephritis, especially the latter. It is more proper to say that during the progress of interstitial nephritis the amyloid degeneration comes on; hence the lardaceous or amyloid kidney may be more or less granular and contracted, instead of being enlarged and smooth. With lardaceous kidney are associated chronic ulceration of the lungs, and suppurating cavities, ulcerations of the intestines, diseases of bones and joints, syphilitic lesions of the mucous membrane, external integument, and scrofulous abscesses.

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* "Lancet," March 13, 1875.

† Beale on "Urinary Deposits."

Symptoms.—As amyloid disease of the kidney arises during the course of some chronic wasting malady, its onset is necessarily obscured by the complexus of symptoms already prominent. There is, of course, a marked degree of anæmia produced by prolonged suppuration, and by amyloid changes in other organs besides the kidney. The urine is, as a rule, increased in amount and may be considerably so, especially in those cases complicated by interstitial nephritis, or it may be considerably diminished in quantity, when there coexists parenchymatous nephritis. But in genuine amyloid kidney the urine is increased, is pale, watery, and of very low specific gravity—1002 not unfrequently—and usually under 1006. When associated with parenchymatous nephritis the specific gravity may rise to 1030, or when, as may happen, the quantity passed is very low. The amount of urea and other solid constituents is much reduced when the quantity is great, and greater when the quantity of urine is small. The amount of urea excreted depends on two factors: on the functional activity of the liver and the extent of disease in the kidneys. Albumin is always present. At times, during the first implication of the kidneys in the morbid process, there may be none, and when present the quantity is sufficient to impart a faint cloudiness merely, but it becomes permanent as a constituent of the urine during the height of the disease, unless just at the close, when it may disappear again. The urine contains so little else than water that the sediment is very small in amount, and hence it requires a good deal of urine to collect even a few casts. Only the hyaline casts are proper to this disease; they are perfectly transparent, homogeneous, and slender, so that they are seen only by careful management of the light. Large granular casts, blood-corpuscles, and renal epithelium may be present in considerable quantity when parenchymatous nephritis is a complication. The casts may present a faintly yellow and highly refracting appearance when attacked by the amyloid change or composed of the amyloid material.

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* Safranine, an aniline product, is said to be an admirable test for amyloid matter. Sections are immersed in a very dilute watery solution. The amyloid matter is stained orange-yellow; the rest of the tissue, rose.

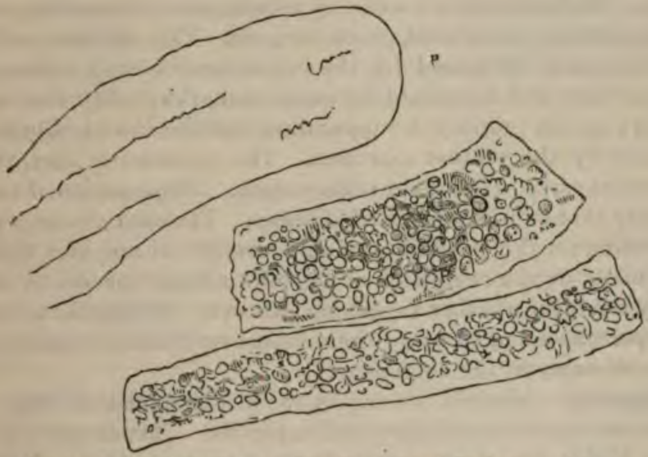

FIG. 41.—A Large Hyaline Cast without, and Two with Epithelium. (Beale.)

More or less œdema is always present, but general dropsy is infrequent. The œdema is found in the lower extremities, and ascites is usually present, and disproportionate to the quantity of fluid elsewhere. This is doubtless due to the implication of the liver in the general morbid process, and to the swelling of the lymphatics in the hilus of the liver, compressing the vena porta. With the progress of the disease, there are necessarily increasing weakness and anæmia, a peculiar earthy or fawn color of the skin, and pigmentation of the eyelids. The exhaustion of the vital forces is greatly hastened by the occurrence of a profuse, watery, and uncontrollable diarrhœa. Vomiting also occasionally takes place, but not nearly with the frequency and persistence of the diarrhœa.

Course, Duration, and Termination.—Amyloid kidney is an essentially chronic malady, but its fortunes partake of the changes and progress of the associated malady. Commencing insidiously, its presence is recognized only when an increasing urinary discharge calls attention to the state of the kidneys. The duration of the disease is largely determined by the suppurating malady causing it; but, when the amyloid change is clearly begun, the case usually terminates in death in a few months, but may extend to years. Uræmia, as manifest in vomiting, purging, amaurosis, partial and general convulsions, etc., does not occur in amyloid disease, unless the contracting kidney also develops, or there is a sudden appearance of parenchymatous nephritis. Death by cerebral hæmorrhage is also rare. Hypertrophy of the heart and of the arterioles does not take place in this form of kidney-disease. The termination is often by some acute inflammation, as pneumonia, pleuritis, or purulent peritonitis, etc. The duration will necessarily be much influenced by the occurrence of such inflammation. Many of the cases terminate by exhaustion, the bodily forces being worn out by the protracted suppuration and the loss of albumin, but especially by the profuse diarrhœa. The termination may, then, be due in most cases to lesions of other organs. The question of recovery is largely that of the associated diseases. The data do not yet exist for deciding on the possibility of an arrest of the amyloid change in the kidneys, or the regression of deposits already made, but it is extremely doubtful whether a genuine case ever terminates in recovery. In a reported case of recovery there must ever remain a doubt respecting the accuracy of the diagnosis.

Diagnosis.—Amyloid kidney is to be distinguished from parenchymatous nephritis and interstitial nephritis. The history of the case is here highly important, especially the constant relation of suppuration to lardaceous degeneration. In parenchymatous nephritis the urine is scanty, high-colored, of high specific gravity, and deposits an abundant sediment, containing urates, granular casts, tubular epithelium, and red-blood globules; in amyloid kidney the urine is abundant, pale, of low specific gravity, deposits very little sediment, containing a few hyaline casts and occasional waxy casts, but no blood-corpuscles. In parenchymatous nephritis, dropsy forms quickly and is extensive; in amyloid kidney, the effusion is slight and confined to the lower extremities and to the peritoneal cavity. Amyloid kidney is distinguished from chronic interstitial nephritis by its history and association with suppuration in some form, and with the evidences of the same change in the liver, spleen, and intestinal canal. In chronic interstitial nephritis the symptoms of uræmia are very pronounced at some period; in amyloid kidney these symptoms very rarely occur at any period.

Treatment.—As when the amyloid deposits have taken place it seems doubtful if their removal can be effected, it is highly important to stop all sources of suppuration, and thus prevent the deposition of the altered fibrin. Attention should be directed at once to the cure of suppuration. As syphilis and the suppuration connected with it are a fruitful source of mischief in this direction, this malady should be efficiently treated and cured, and all cases presenting a syphilitic history should be given a thorough course of the iodide of potassium.

Dickinson, influenced by his theoretical notions, advises the internal use of the potash and soda salts, supplying artificially the alkali which is carried off in the pus, while the fibrin is deprived of it. He at the same time enjoins the free use of eggs and milk, to supply the material lost in the urine. A combination of the chloride of gold and sodium ($\frac{1}{16}$ gr.) and bichloride of mercury ($\frac{1}{30}$ gr.), persistently administered, accomplishes more than any other remedies. Iron, cod-liver oil, and a generous diet are demanded by the condition of feebleness and anæmia. The exhausting diarrhœa resists all means of treatment, but the most efficient remedy, according to the author's experience, is Fowler's solution and opium tincture—three drops of the former and five to ten of the latter, three or four times a day.

PYELITIS AND PYELONEPHRITIS.

Definition.—*Pyelitis* means an inflammation of the pelvis of the kidney; *pyelonephritis* includes pyelitis and a consecutive or simultaneous suppurative inflammation of the kidneys. They are here considered together to avoid repetition, and because of their frequent association.

Causes.—Probably the most frequent cause of pyelitis is the extension of a morbid process from the bladder to the pelvis of the kidney, by the ureter. Catarrh of the bladder is lighted up by decomposition of the urine, consequent on its retention. Whenever an obstacle exists to the discharge of urine from the bladder, the decomposition ensues, the urine becomes ammoniacal, and the mucous membrane the seat of an active catarrhal process. Stricture of the urethra, enlarged prostate, the pressure of the retroverted uterus, pregnant uterus, or of a pelvic tumor, etc., act by hindering the urinary discharge. An inflammation of the mucous membrane of the bladder, due to gonorrhœa or other causes, will have the same effect by causing fermentation of the urine. A renal calculus, or other foreign body, present in the pelvis of the kidney, will produce catarrh directly by irritating the mucous membrane. Decomposition of the urine and catarrh extending to the pelvis of the kidney are produced by paraplegia: the bladder being paralyzed, the urine is retained and undergoes putrefactive fermentation. Diuretics of the stimulant kind, as copaiba, turpentine, and cantharides, irritate the mucous membrane of the pelvis of the kidney in passing through these organs. Whenever the urine decomposes, vibrios and bacteria appear in it in immense numbers; the urea is decomposed and converted into the carbonate of ammonia; the ammoniaco-magnesian phosphate crystals are formed in great quantity, and much phosphate of lime is separated by the inflamed mucous membrane. Pyelitis occurs as a complication in various infective maladies—in pyæmia, puerperal fever, the exanthemata, etc., and may result from the extension of a neighboring inflammation.

Pathological Anatomy.—The changes consist in the ordinary catarrhal process, the mucosa and the submucosa becoming very much thickened in old cases, the vessels varicose, and the epithelium much changed by the proliferation of its cells, etc. If the morbid process began in the bladder, the evidence will be plain, and the ureters may or may not be affected by the same changes. If the pyelitis has existed for some time, the kidneys will be seen to be in a process of suppuration—one or both. The organ is more or less enlarged, is deeply congested and reddish, except certain spots which present a yellowish-white color, are wedge-shaped, and extend through the cortex to the apex of the cone. On section these patches present here and there points of suppuration, are swollen, and the capsule is more or less firmly adherent to them. Suppuration occurs soon all along the extent of these patches between the tubules. Several of these suppurating patches uniting, considerable abscesses form ; the kidney elements are disassociated, broken up, and disappear ; and from the cones the suppuration proceeding destroys the cortical part, and ultimately nothing remains but a bag of pus having irregular walls marked by septa, remains of calyces. It seems well established that the suppurative inflammation in the kidneys is set up by the presence of bacterian colonies which have migrated from the inflamed bladder. With high powers the bacteria are seen arranged in parallel lines within the tubules. They appear as minute, globular, highly refracting granules. After a time the same bodies are seen in the interstices with pus-corpuscles. The epithelium of the tubules is at first cloudy, granular from fatty degeneration, but is soon destroyed, the whole tube being filled with the branching filaments and spores. According to Klebs (Ebstein), the inflammation proceeding to suppuration is excited by the bacteria.

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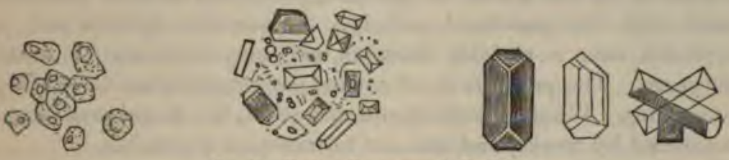
FIG. 42.—Various Forms seen in Pyelitis.

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Diagnosis.—In the most common variety the diagnosis is often merely conjectural, for the muco-pus is so abundant that it is extremely difficult to find the characteristic forms from the pelvis. When uræmic symptoms finally come on, there can be doubt no longer. In the simple cases the diagnosis must rest on the association of pain, with altered urinary secretion, the epithelium of the pelvis of the kidney being present.

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* London "Lancet," December 5, 1874.

Pathogeny.—The researches of Dr. H. Vandyke Carter, Ord,* Beale, and others have demonstrated the importance of mucus in determining the precipitation of the calculous ingredients of the urine. Calculi are of all sizes—from microscopic bodies up to a concretion filling the pelvis of the kidney. Beale † has shown the importance of microscopic calculi present in the urine, as indicating similar bodies of larger size in the pelvis. In the kidneys there may be an infinitude of calculi—from mere grains of sand to concretions of considerable size. Uric-acid infarctions, triple phosphate- and carbonate-of-lime infarctions, are found in the straight tubes of the pyramids in infants, and in old men, especially those affected with the gouty diathesis. Calculi of uric acid are more frequent than any other constituent, for, although this substance exists in small quantity, it is very slightly soluble. Ord shows that uric acid, crystallizing in the presence of colloids (albumin, mucus, etc.), tends to assume a spheroidal form, and Carter that a bit of mucus is the nucleus about which the crystallization takes place. These calculi are made up of concentric layers, and may be composed wholly of uric acid, or of alternate layers of uric acid and oxalate of lime. Similar modifications are impressed on oxalate of lime, but while they tend to assume the spheriodal form in the presence of mucus they also crystallize in octohedra. The uric-acid calculi are grayish-red or reddish-brown, smooth, hard, and having a specific gravity of 1·5. The pure oxalate-of-lime calculi are very rare, are very hard in texture, rough on the exterior, of a dark-brownish color. The oxalate of lime with a nucleus of uric acid are much more common than the pure oxalate. Calculi of cystine are still more rare than those of oxalate of lime; they are comparatively soft, and have a dull-yellow or amber color. Phosphatic, next to uric, are the most frequently encountered calculi. They are very light, friable, of a dull or grayish-white, or bright white, rough, and sometimes polished. The phosphatic deposit, consisting of phosphate of lime and the ammoniaco-magnesian phosphate, often forms about a uric-acid calculus which has been present for some time. This deposition of the phosphates may be expected to take place on a uric-acid calculus which has been long present in the pelvis of the kidney, if the urine becomes alkaline. The stones may be in one, but occasionally they are found in both kidneys. In the cases which have fallen under my observation, two thirds were in the left kidney. The results of the presence of concretions differ according to their situation: in the tubules, as infarctions, they excite inflammation of the kidney; in the pelvis they cause pyelitis. Gouty kidney is a result of the uric-acid diathesis, and deposits of this substance take place in the pyramids and the cortex, parenchymatous and interstitial nephritis develop, and the organs ultimately become granular. When nephropyelitis is fully developed, extension of the morbid process to the kidney proper takes place. When pyelitis is lighted up, the mucous membrane becomes intensely injected, and a quantity of muco-pus, proliferating epithelium, and young cells, form a yellowish, rather thick, puriform fluid. If a concretion is not too large, it will be washed down into the bladder, with the phenomena of nephritic colic. Successive calculi passing, the ureter yields and dilates, and, as these concretions, in passing, excite inflammation, the walls of the ureters become thickened. An attack of inflammation may close the canal entirely, or a ureter may be closed by an impacted calculus. In either case the contents of the pelvis accumulate, the proper structure of the kidney undergoes atrophy, and after a time only a membranous sac filled with fluid and concretions remains. The ichorous contents may ulcerate through, form an abscess of large dimensions, which may make its way externally, discharging in the lumbar region, or, dissecting downward, may point underneath Poupart's ligament, or enter the colon, etc.

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* "Lancet," March 13, 1875.

† Beale on "Urinary Deposits."

Symptoms.—A calculus may remain in the pelvis of a kidney for a long time—during many years—it is probable, without giving rise to any disturbance. Usually, very distinct symptoms are occasioned, and serious results grow out of them. A calculus causes very violent symptoms when washed into the ureter. Usually, an attack of nephritic colic occurs suddenly. Without any warning, an atrocious pain strikes the lumbar region, passes downward along the course of the ureter into the groin, and radiates thence upward into the shoulder-blade and through the abdomen. Pains occur in the corresponding testis, which is retracted close up to the external ring, and more or less pain, sometimes very acute pain, is felt in the glans penis. So severe is the pain that the most self-controlled person cries out with the agony, rolls from side to side, or rushes up and down the room seeking for some alleviation in incessant motion. The face is pale and torn with agony, the features are pinched, the body is cold and covered with a cold sweat. The thigh of the affected side is benumbed, and sometimes the whole of the corresponding limb. The patient may faint, or pass into unconsciousness with a general convulsion. The stomach participates in the disturbance with nausea, or with severe vomiting. The bladder is very irritable, and frequent attempts at micturition are made, but, with much burning pain and straining, only a few drops are passed. The urine is dark, and usually contains blood, but it may be perfectly normal, for, as but one ureter is involved at one time, the urine from the unaffected kidney may pass without admixture. The urine may be not only dark and bloody, but it may contain pus. There may be complete anuria from blocking of both ureters, but usually the calculi do not fit accurately, and some urine escapes alongside them. If anuria is the result, and the obstacle is not removed, death in coma and convulsions is inevitable. The paroxysm, after some minutes or hours, usually terminates suddenly by the escape of the stone into the bladder. The urine accumulating behind the stone forces it onward with increasing agony, until, at last dropping into the bladder, the horrible pain ceases, the patient utters a sigh of relief, and falling on the bed exhausted is soon fast asleep. The attacks do not always come on abruptly. There may be experienced some deep-seated soreness in the lumbar region, then a quick movement as in kicking, sneezing, coughing, etc., may give rise to a sudden increase of the soreness, soon developing intō acute pain. Whether the onset be sudden

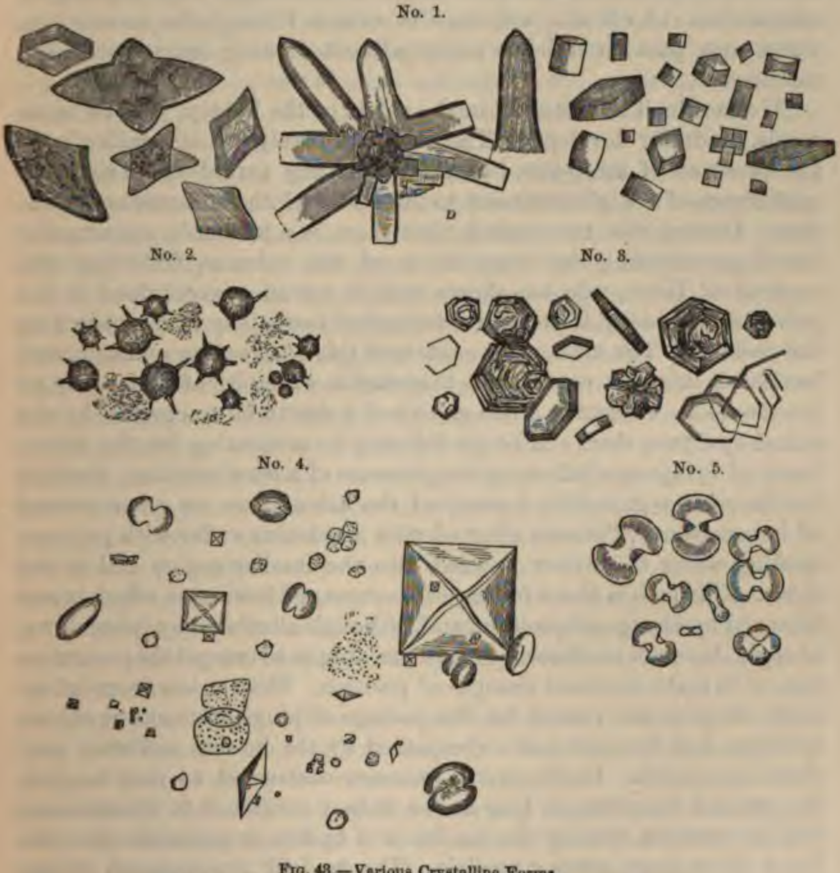

Fig. 43.—Various Crystalline Forms.
No. 1.—Uric Acid. No. 2.—Urate of Soda.
No. 3.—Cystine. No. 4.—Oxalate of Lime.
No. 5.—Dumb-bell Oxalate of Lime.

or gradual, the attacks are not of equal severity. The difference we may suppose to be due to the varying sizes of the calculi. If a calculus become impacted, it will ulcerate through and give rise to fatal peritonitis. In a few cases the calculus has occupied a number of days in making the journey through the ureter, the most severe suffering, as is usual, occurring at last, owing to the increasing narrowness of the lower ureter. If repeated attacks occur, the rule is that the succeeding ones are milder, but this depends upon the size of the calculi. Gravel and sand may occasion no distress at all, or, at most, some little burning at micturition. A calculus too large to escape through the meatus urinarius may pass through the ureter without causing recognizable disturbances.

If the calculi are retained in the pelvis of the kidney, pyelitis is, as a rule, gradually developed. The urine ultimately becomes milky from the presence of muco-pus, but there is a long period from the first appearance of a slight sediment to the milky-white appearance on emission. During this intervening time there is a favorable opportunity for diagnosticating the composition of the calculus, following the method of Beale, who has shown that, if calculi are contained in the pelvis of the kidney, identical microscopical forms may be recognized in the sediment. The author has confirmed this observation of Beale, and has had in his own cases some remarkable examples of the utility of the method. Of course, when calculi of a size to be recognized by the naked eye pass, there can be no difficulty in accounting for the occurrence of symptoms indicating the presence of a renal calculus. Besides the knowledge gained by a study of the urine, there are other sources of information. Patients affected with a calculus suffer with pain extending along the ureter upward into the lumbar region and to the spine. This pain is also a feeling of soreness and heaviness, which is not removed by change of position, and, although alleviated by lying down at night, becomes so irksome toward morning as to compel the patient to rise, or to make incessant changes of position. More or less frequent attacks of colic are caused by the passage of plugs of mucus or shreds of tissue, but they are not accompanied by the intense suffering produced by calculi. If the ureter becomes obstructed, as may happen, the pus and the urine, so long as the kidney continues to functionate, will accumulate, causing the condition of hydro- or pyonephrosis—the latter when there exists a pyelitis. The gradual accumulation of pus and the disintegration of the kidney substance will result in the formation of a sac with thick walls, presenting evidences of renal structure only on careful inspection. A tumor will form of considerable volume, projecting downward from the hypochondrium. It may be somewhat nodular, irregular, but is more frequently smooth and globular—the outline and shape being determined by the degree of accumulation; hence the tumor is the more globular and less nodular the more ancient. The tumor may attain to very large size; in a case in the author's charge, it was as large as a child's head. The sac may yield and the contents escape into the peritoneal cavity, or a communication may be established with the colon or stomach, or discharging posteriorly may open a fistulous communication in the lumbar region, or dissecting downward along the course of the psoas muscle may point under Poupart's ligament. The calculus may be discharged by any of these channels. When the ureter is closed, the urine, which before was full of pus, now appears clear again. An obstruction of the ureter may be temporary, and the urine after a short period of freedom from pus may become loaded with it again. When the obstruction yields, a sudden gush of purulent urine and *débris* will cause more or less pain or colic; indeed, the attack may have all the characteristics of a severe nephritic colic.

Course, Duration, and Termination.—Nephrolithiasis develops slowly, is very chronic in its course, and variable in the results. The exceptions to this statement consist of those cases which terminate suddenly by rupture of the ureter and peritonitis, and the very rare examples of septicæmia or pyæmia occurring with the beginning suppuration, or of uræmia from the simultaneous blocking of both ureters. Renal sand and small concretions may, after a variable period of detention, pass down the ureter and be discharged with the urine. Often concretions of considerable size, too large to pass the meatus urinarius, are thus discharged, all symptoms ceasing when the source of irritation is removed. Recovery has ensued also by the discharge of the concretion through a fistulous communication externally, the kidney undergoing atrophy, the sac closing, and the formation of pus ceasing. As one kidney may perform the duty of both, a cure effected in this way may be genuine. Death may occur from exhaustion, or amyloid degeneration may be the result of the protracted suppuration; pyæmia, or some intercurrent malady, may quickly terminate life in a portion of the cases.

Diagnosis.—Renal colic may be confounded with biliary colic. The two affections are frequently associated. They are distinguished by the situation of the point of maximum pain, and by the sequelæ—hepatic colic followed by jaundice and pasty stools, renal colic by excessively irritable bladder and bloody urine. Is the calculus present uric or phosphatic? The preponderance of numbers is a presumption in favor of uric acid. But the determination is made by an examination of the sand, gravel, or microscopic calculi. A uric-acid calculus, long present in a suppurating pelvis of the kidney, will become more or less deeply incrusted with phosphatic material, and the urine will contain phosphate crystals. When a tumor exists, the kidney affected is revealed. That one and not both kidneys is the seat of disease may be determined by the passage of perfectly normal urine when an obstruction, either temporary or permanent, prevents the escape of pus.

Treatment.—As the attack of renal colic requires the most powerful anodynes, morphine hypodermatically should be employed at once. As the stomach is highly irritable, it is useless to give medicines by the mouth for this purpose. Enemata of laudanum act efficiently if sufficient time be given them. The inhalation of ether may be practiced until more permanent relief can be given. The warm bath is serviceable by inducing relaxation. If gravel or sand of uric acid is present, its solution and excretion should be effected as speedily as possible. The urine should be alkalinized by the free use of the potash and lithia salts; soda must be avoided, as the urate of soda is not readily soluble. Probably the best preparation is the officinal *liquor potassii citratis*, of which a tablespoonful may be taken every three hours. Recently the borocitrate of magnesium and the benzoate of lithium * have been used successfully, both of these agents having remarkable solvent effects on uric-acid calculus. The experiments of Roberts,† however, seem conclusive as to the solvent action of the potash salts; these failing, the borates and benzoates may be tried. Nothing can be accomplished by spasmodic efforts. The solvent action must be maintained without intermission for a long period. Should the protracted existence of a uric-acid calculus, with pyonephritis and alkaline urine, render it probable that an incrustation of phosphates has occurred, the benzoate of ammonia should be prescribed, as the most certain means of bringing about an acid condition of the urine. If the calculus is phosphatic, the same procedure is proper to produce and maintain an acid state of the urine until the phosphatic incrustation or the phosphatic calculus is dissolved. When this is accomplished, the method above mentioned must now be pursued. In the treatment of pyelitis those remedies are to be employed which are eliminated by the kidneys and exert a local action—copaiba, cubebs, santalum, juniper, erigeron, eucalyptol, turpentine, etc. These must be used with caution, because of their irritant effects on the kidneys. Probably the most generally useful, and at the same time safe, is eucalyptol. This should be administered in small doses, relying upon the results of a slight impression maintained for a long time. Any of the members of this group may be employed instead of eucalyptol, under the same limitations. The so-called diuretics—scoparius, squill, buchu, pareira, etc.—have also been recommended, but they are less effective than the oils. Cantharides tincture has been prescribed in small doses with advantage in pyelitis. The free use of skimmed milk, and buttermilk when it is grateful or preferred, is decidedly beneficial. When the existence of the tumor can be made out clearly, it should be evacuated posteriorly by the aspirator. If the calculus can be reached, a free opening should be made and a drainage-tube inserted. The sac can then be kept thoroughly empty, clean, and in the most favorable condition for shrinking and ultimate closure. Recovery has ensued. In a case of the author's in which the sac was opened from behind, the calculus was removed and free drainage secured, but the patient was exhausted by protracted suppuration.

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* "Bulletin Général de Thérapeutique," January 30, 1880.

† "Urinary and Renal Diseases," *op. cit.*

HYDRONEPHROSIS—DROPSY OF THE KIDNEY.

Definition.—*Hydronephrosis* consists in an accumulation of the urine and dilatation of the pelvis and calyces, with progressive atrophy of the renal structure.

Causes.—Hydronephrosis may be congenital or acquired. When congenital it is due to some anatomical anomaly. It is more common in women than in men, because of the functions peculiar to the former. Obstruction of the ureter is the usual cause ; the nature of the obstruction may differ greatly. The ureter may be blocked by a calculus, by inflammation and adhesion of the mucous surfaces, by constriction of a band of lymph, by pressure of a tumor, by the displaced uterus, etc. When an obstruction is caused by the impaction of a calculus, it is usually found *in situ ;* but not invariably so, for sometimes the calculus crumbles and disappears.

Pathological Anatomy.—The dilatation will involve the more of the ureter, the lower down the obstruction is placed. The degree of damage done to the kidney will, of course, be determined by the amount of fluid. In an extreme case the kidney-structure will have disappeared, nothing remaining but a huge membranous bag, the ureters distended into somewhat tortuous cylinders the size of a small intestine, and with more or less thickened walls. When the accumulation is small in amount, the pelvis is somewhat dilated, the calyces also, and the papillæ are flattened. As the fluid increases, there will be increasing atrophy of the kidney, the medullary portion first disappearing, and ultimately the cortical part. The sac may be of enormous dimensions, filling half the abdominal cavity, displacing organs and contracting adhesions to neighboring parts. The colon may be compressed and adherent very closely to the walls of the sac. The original capsule of the kidney, thickened by new connective tissue, forms the walls of the sac, the lobulated appearance being due to the internal septa. The fluid in the sac is modified urine—it is pale, of low specific gravity, alkaline in reaction, and contains urea, uric acid, urates, etc., or it may be brownish in color from the presence of

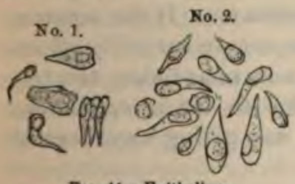

FIG. 44.—Epithelium.
No. 1, Ureter. No. 2, Urethra.

blood, or yellowish and turbid from the presence of pus (pyonephritis). The fluid usually contains traces of albumin, which may be considerable if blood is present, and more or less epithelium may also be occasionally found. The accumulation is usually limited to one kidney, the other being enlarged to compensate for the absence of its fellow.

Symptoms.—It is an extremely rare event for both kidneys to be affected, and hence uræmia is not a common, is indeed a rare symptom. The accumulation occurs silently, and hence it is the formation of a fluctuating tumor that first attracts attention. The size depends somewhat on the age of the growth; it may have the dimensions of a child's head. In growing, adhesions form, which give rise to acute, stabbing pains at the time of their formation. When the tumor attains sufficient volume to displace or compress the neighboring organs, corresponding disturbances are occasioned. If the colon is compressed, great accumulation of fæces will take place above that point; if the diaphragm is pushed up, dyspnœa will result; if the stomach is pressed on, there will be nausea and vomiting; if the tumor rests on the abdominal aorta, a pulsation will be communicated to it. It is important to note that the colon in hydronephrosis of the left side may lie in front of the tumor. The author saw a surgeon pass a trocar through the large intestine to reach the sac! The tumor has usually some firmness, does not fluctuate very easily, although distinctly, and is not movable. It may be handled freely without pain, as a rule, unless adhesions have recently formed, when it will be tender.

Course, Duration, and Termination.—The course of hydronephrosis is chronic, the onset obscure, the formation of a tumor slow, and the final disposition of the sac a tedious process. Years will be occupied in the development of these several stages. A genuine cure is rarely effected. It may happen that an obstruction within the ureter yields and the water flows away, but this is very uncommon. If the accumulation be due to pressure of a displaced uterus, a cure is readily effected by correcting the displacement. When not remediable, the termination is ultimately fatal, death being due to the complications arising from the pressure on organs, or, the sac giving way, general peritonitis is the result.

Diagnosis.—Hydronephrosis is most frequently confounded with ovarian tumor. The former develops from above, the latter from below. The withdrawal and examination of the fluid are usually necessary to come to right conclusions. The fluid of hydronephrosis is usually watery and contains urea, uric acid, and epithelium; the fluid of ovarian disease contains the compound, granular, many-nucleated corpuscles, is dark in color, and somewhat gelatinous in consistence. Hydronephrosis may be confounded with ascites when both kidneys are affected. They are to be distinguished by the changes in the position of the dullness, on changes of posture in cases of ascites, which do not occur in hydronephrosis. In the beginning of ascites, if the patient lies recumbent, the dullness is in the flank ; in hydronephrosis the dullness is at the site of the tumor, and does not change its position.

Treatment.—The sac has been emptied by careful manipulation, the obstruction yielding to pressure. This treatment is applicable but rarely. If the accumulation is sufficient to endanger life, the aspirator may be used, but otherwise interference is to be deprecated.

CARCINOMA OF THE KIDNEY.

Causes.—Nothing is definitely known of the causes of cancer of the kidney. It may be primary or secondary. It occurs in early life —before five—and in old age, youth and manhood to middle age being comparatively exempt. As regards sex, cancer of the kidneys is more common in men.

Pathological Anatomy.—Primary cancer rarely involves both kidneys, and of the two the right is the more frequently attacked. When cancer of the kidney is secondary, the organ attacked by contiguity, one only is affected, but, if there exists a general carcinoma, both will be the seat of deposits. The cancerous kidney attains to great size—according to Rindfleisch to twelve inches in length and six inches in width, and to a weight of sixteen pounds (Spencer Wells). This enormous size is attained in a very short time. Again, although very rarely, the kidney may not be enlarged by cancer deposits. The shape of the organ may be exactly preserved, or there may be irregularities and nodosities ; in the former the organ on section presents a uniform whitish or yellowish surface ; in the latter the cancer-masses occur in distinct nodules, separated by a defined line from the normal tissues, or become encapsulated. The vessels of renal cancer are abundant, large, and have thin walls, are consequently easily ruptured, the blood collecting in large excavations. Usually there is very considerable hyperæmia of the interstitial connective tissue, which assumes an active hyperplasia. Sometimes there is found in the midst of a mass an isolated soft detritus, made up of cells which have undergone fatty degeneration and may have a foul odor. The cancer elements, according to Waldeyer,* whose views are accepted by Rindfleisch,† develop from the epithelium of the tubules. The form taken by the cancer is determined by the relative proportion of fibrous stroma and cells and blood-vessels. The more abundant the vessels and cells, the softer and more rapidly growing the cancer, which is then called the medullary carcinoma. If the fibrous framework or stroma is in excess, then the cancer becomes scirrhous. The cancer may spread to and involve the pelvis and ureter, and the latter may be filled up with cancer-masses. The pelvis may be filled with blood-clots, stratified as in aneurism. The cancer elements may invade the renal vein, reach into the *vena cava* by coagula, whence emboli are detached, and lodge in the lungs. The cancerous kidney may contract adhesions to adjacent parts, and is apt to do so, or, detached by its increased weight, may become migratory or floating. If it remains in its own position and enlarges to the enormous extent that sometimes occurs, neighboring organs may be much displaced and compressed, those of the thorax as well as those of the abdomen.

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* Virchow's "Archiv," *op. cit.*

† "Pathological Anatomy," p. 512.

Symptoms.—Cancer of the kidney may develop to a considerable extent without producing any characteristic symptoms. Pain may be experienced to a greater or less degree in the beginning, but it does not differ from pain due to other causes. It is felt in the lumbar region, under the false ribs, external to the spine, and is a sensation of soreness merely, rather than the acute, lancinating pain traditional of cancer. With or without pain, hæmaturia occurs, and is the first symptom to awaken a suspicion of the nature of the malady, but this symptom is present in one half of the cases only. It is not constant, and there may be considerable intervals of a few days, weeks, or months between the hæmorrhages. Its appearance may be postponed until near the end. It not unfrequently happens that some external injury, a blow, a fall, determines the hæmorrhage or increases its violence. Rarely is the quantity of blood sufficient to cause dangerous exhaustion. The urine may present a faint, smoky hue; it may be reddish or reddish-brown; it may contain clots of various sizes. The corpuscles are more or less crenated and otherwise altered when the urine is merely smoky, but when the quantity of blood is considerable the corpuscular elements are normal. So long as the blood is intimately mixed with the urine, there is no pain connected with it; but, when clots of considerable size are forced through the ureter, the pain will be agonizing—only less severe than that due to the passage of a calculus. Although in the beginning there may be only some deep-seated soreness, or no pain of any kind, in the further progress of the case pains will come on. The pain may be deep and rather dull in the neighborhood of the kidney or in the lumbar region, or it may be sharp, lancinating, and radiate along the intercostal nerves, or downward into the hip, the whole of the corresponding lower limb feeling benumbed and heavy. Sometimes excruciating sufferings are experienced in the sciatic nerve by pressure of cancerous lymphatics, and the limb rapidly wastes. Sufficient enlargement of the kidney to constitute a tumor is the most constant symptom. In sixty-four cases a tumor of the abdomen was recognized in all but three, and in nearly all of these was of a size to be recognized on a cursory examination

(Roberts). The tumor pushes forward into the anterior part of the lumbar region and grows upward into the hypochondrium and downward toward the iliac regions. In children the tumor attains the largest growth, filling the entire abdomen. As the colon lies in front usually, and as the material of the cancerous kidney is soft, the tumor does not furnish a dull or flat note on percussion, but a distinctly tympanitic note. Full inspiration or expiration does not affect the position of the tumor, which is usually, but not invariably, immovable; the cancerous kidney may also be a movable or a floating kidney. By carefully relaxing the abdominal muscles the form and density of the tumor may be ascertained. It will be found somewhat elastic, round, and smooth, or hard, firm, and nodular. There may be a ramification of enlarged veins on the abdominal surface of the tumor, and it may have a pulsation in it, communicated from the abdominal aorta. If hæmaturia is absent, the urine may be normal in amount and quality. It occasionally happens that albumin is present when there is no blood, because of a coincident Bright's disease. Uræmia does not occur because the disease is unilateral, but both organs may be involved. When a calculus is present, as is not unusual, pyelitis will complicate the renal symptoms. Particles of broken-down tissue and the so-called cancer-cells are sometimes to be found in the urine, but unfortunately there is no distinctive cancer-cell. The digestion may be unimpaired, the appetite keen, even voracious, but the rule is that the appetite is poor, there is nausea, and the body wastes. With the first symptoms there is emaciation, which ultimately becomes extreme.

Course, Duration, and Termination.—This disease does not pursue the same course in all cases. In children the progress is more rapid, the mean duration being seven months (Roberts), whereas in adults the average duration was two and a half years. In some cases in children the duration is counted by weeks, and one case is noted as occurring in an adult which lasted eighteen years. The termination is invariably in death. Sometimes unexpected improvement takes place, but evil symptoms come on again presently.

Diagnosis.—Cancer of the right kidney may be mistaken for a tumor of the liver. It is usually possible to demonstrate a sulcus between the liver and the enlarged kidney, or to insinuate the fingers between the two. The position of the colon is an important element, for lying in front of the kidney modifies the percussion-note, which is *dull-tympanitic* over the kidney and flat over the liver. From an enlarged spleen it is to be distinguished by the evolution and position of the tumor; by the situation of the colon—in front of the renal and behind the splenic tumor; by the shape and thickness of the tumor—the spleen having a rounded margin and comparatively thin edge which may be grasped; by the history of the case—malaria or leucocythemia of a splenic tumor; by the urine, containing blood and cancer elements, etc. From ovarian tumor the differentiation is made by the position of the growth, the mode of its development, by its form; by the position of the colon, again; by the occurrence of hæmaturia, etc. From accumulations in the large intestine, in the cæcum—the descending colon—the kidney-tumor is recognized by its size, outline, position, and percussion, by hæmaturia, by the action of a cathartic, or irrigation of the bowel. As cancerous tumors of the kidney sometimes pulsate, they may be mistaken for aneurism. If the patient be placed on the elbows and knees, so that the tumor may glide away from the aorta on which it lies, the pulsation will cease. If a fixed tumor, this expedient can not be practiced. A fixed tumor of that kind pulsating, will produce no expansile movement. It will be very confusing if a minute communication exist between the aneurism and pelvis of the kidney, for then hæmaturia will coexist with a tumor.

Treatment.—The remedial management is merely symptomatic, and is chiefly confined to measures for the relief of pain.

TUBERCULOSIS OF THE KIDNEY.

Pathogeny.—The deposit of tubercle occurs in the two forms—disseminated, localized. In the disseminated form, gray granulations are scattered through the renal parenchyma, and are developed from the sheaths of the vessels, and this form is a part of a general morbid change. It is the localized form with which we are chiefly concerned here. The deposit of tubercle-masses begins at the renal papilla by an extension of the morbid process taking place in the calyces and pelvis. The miliary nodules aggregating, assume the cheesy aspect, soften in the center, are extruded, carrying with them the portion of tissue embraced in the deposit. Thus an excavation is established. The kidney usually increases somewhat in size; it becomes nodular, and the capsule, thickened and indurated, contains various foci of cheesy deposit. The whole organ is ultimately converted into a mere bag with thick walls and projections inwardly of connective-tissue septa, the remains of the original calyces. The testes and epididymis are, in the majority of instances, the seat of the initial changes, and spread thence to the kidneys, or they may begin in the bladder and extend thence into the kidneys. The same cheesy infiltration takes place in the pelvis, ureters, and bladder.

Symptoms.—The urine is increased in amount, and contains, when the disease is developed fully, blood and pus, the reaction is acid, and albumin is present. In the further progress of the case, the urine becomes ammoniacal, alkaline, and thick with pus and detritus. When the disease has reached the sub-mucous tissue, shreds of elastic tissue and fragments of cheesy matter, with the bacillus tuberculosis, which indicate clearly the nature of the destructive changes, appear in the urine. Micturition is frequent and more or less painful. This is due to the tubercular ulceration of the mucous membrane of the bladder, and the catarrh which accompanies it. More or less pain is experienced in the lumbar region, which may be a feeling of soreness and fatigue combined, or of acute pain, paroxysmal in character. Besides the lumbar pain, there are paroxysmal attacks of pain in the back, extending along the ureter, attended with frequent and painful micturition, produced by the passage of shreds of tissue or cheesy masses. There may be no pain. Obstruction to the ureter taking place, there may ensue an enlargement of the kidney of sufficient size to constitute a tumor. The obstruction yielding, the accumulated pus and urine will flow away, and the tumor will collapse; but when the tumor once forms, although it may vary in size, it does not entirely disappear. With the progress of the tubercular ulceration, there is increasing destruction of the renal substance, and hence the quantity of urine is constantly declining. As both kidneys are usually affected, uræmic symptoms come on, when the excretion of urea and other effete materials is prevented. Usually, however, the patient is carried off by the progress of the tubercular ulceration in the intestinal canal and lungs. In the author's cases, there were simultaneous pulmonary lesions, which, however, seemed to make but little progress. Death occurs by exhaustion, or with some head troubles.

Course, Duration, and Termination.—The course and duration are much affected by the existence of general tuberculosis, by the extent of mischief in both kidneys, and by the degree in which the bladder is implicated. The duration rarely exceeds one year, though there are occasional examples lasting two, even three years. If the bladder is much affected, the pain and irritation and the loss of sleep from frequent micturition rapidly exhaust the vital powers. If both kidneys are largely damaged, the case will be terminated by cerebral hæmorrhage, or by coma and convulsions.

Treatment.—The best results as regards prolongation of life are obtained by the use of quinine in considerable doses (five grains *ter in die*), the benzoates, salicylates, and eucalyptol. To relieve the irritable bladder and permit sleep, the most suitable remedies are chloral and morphine by suppository or enema. If the cystitis is very severe, and the urine ammoniacal, good results are obtained by the author by irrigation of the bladder with a weak solution of salicylic acid and borax.

ECHINOCOCCUS OF THE KIDNEY—HYDATID CYST OF THE KIDNEY.

Definition.—*Echinococcus of the kidney*, like echinococcus of the liver, is the immature or larval condition of the *tænia echinococcus*, the tape-worm of the dog.

Pathogeny.—According to Davaine, this parasite is rarely found in the kidney. It is a sac composed of several layers, transparent and hyaline, the mother-sac, and within it are contained a watery fluid and a number of small vesicles (daughter-vesicles), attached to the brood-capsule (mother-sac) or floating freely. These daughter-vesicles vary in size from a grape-seed to an orange—the largest containing their own progeny, or granddaughter-vesicles. As the daughter-vesicles enlarge, the brood-capsule with its germinating layer also enlarges. Within each capsule or vesicle is seen the scolex, or so-called head with its suckers and row of booklets. The fluid of the vesicles is watery, albuminous, and saline, and contains, besides chloride of sodium, crystals of uric acid, oxalate of lime, triple phosphates, and plates of cholesterine. The parent-vesicle is inclosed in a vascular, white, dense connective-tissue tunic or envelope, one half to two or three lines in thickness, and firmly adherent to the surrounding gland-substance. The size of the cysts varies from a small marble to a child's head, and it is situated in the substance of the kidney, and occasionally between the capsule and the gland-structure. The pressure of the enlarging cyst induces atrophy of the adjacent renal substance, until ultimately the whole organ may be destroyed. Rupture may take place into the pelvis of the kidney, but not into the peritoneum, this untoward result being prevented by a limiting adhesive peritonitis. Adhesions are also formed with neighboring organs. Sometimes the cysts are sterile, and consist of a single cavity. The growth may be arrested and the cyst undergo calcareous degeneration mixed with a fatty change. In two thirds of the cases a rupture of the cyst takes place into the pelvis, and pyelitis is produced thereby.

Symptoms.—As the parasite is deposited in one kidney—a rule to which there are but few exceptions—and as no disturbance is caused in the functions of the affected organ until the parasite has attained a certain development, it is obvious that the first period of growth will escape recognition. As the tumor enlarges, neighboring organs are displaced, and as inflammatory attacks are excited and adhesions formed, these will be accompanied by attacks of pain and feverishness. Usually only the symptom of a tumor, smooth and elastic, in the flank, is experienced, and for which advice is sought. According to Roberts's statistics, of sixty-three cases of hydatids of the kidney, only eighteen presented the symptom of a tumor, varying in size from an orange to an adult's head.* Fluctuation was distinct in part, feeble in others, and not perceived in the rest. The most characteristic symptom is the "purring tremor," "the hydatid thrill," unfortunately a symptom which is not often encountered. It can be produced only when there are numerous daughter-vesicles inclosed in the mother-sac, the thrill being caused by the collision of these elastic bodies. The tumor will usually have the colon in front, but it may be at one side. The dullness on percussion will be much influenced by the position of the bowel, which must always be taken into consideration. As the cyst in a majority of cases tends to rupture into the pelvis of the kidney, the symptoms connected with this are of great importance. Vesicles, shreds of the germinating layer, and an emulsion of milky appearance containing fragments, hooklets, and oil-drops, are discharged with the urine, and at once indicate the character of the case, the microscope being used to find the hooklets. The rupture of a sac into the pelvis of the kidney is usually announced by the occurrence of sharp pains in the region of the kidney, with a sensation of something giving way. This seems all the more probable if, as has been the case, the patient has received a blow in the side, followed by the sensation of something giving way. The pain descends by the ureter, the testicle is retracted, the surface cold, and the pulse feeble. The severe attacks of renal colic are comparatively rare, but some pain in the loin and down the ureter is usual. The paroxysms do not continue longer than a few hours, or a day or two, to be resumed again at variable intervals of a few weeks, several months, or even three years. When the vesicles reach the bladder, the pain of renal colic ceases ; but new troubles arise in the attempt to pass these bodies by the urethra. Violent vesical tenesmus comes on, pain is felt at the glans penis, and with severe straining the vesicles are passed, but not unfrequently the aid of the catheter is necessary to empty the bladder. If there be a single mother-vesicle, the discharge of the daughter-progeny may end the symptoms by the shrinking and closure of the sac. The sac may be much reduced in size by the discharge, but fills up again, the same phenomena being repeated from time to time.

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* "Urinary and Renal Diseases," p. 572.

Course, Duration, and Termination.—The course of hydatids of the kidney is chronic, and the duration uncertain. If a single cyst is present, the discharge of its contents may terminate the case, but usually there are several discharges. The cases may terminate by rupture into the peritoneum, which is unusual, by bursting into a bronchus, by exciting pleuritis, by suppuration in the sac, by some intercurrent malady, and by recovery, which occurs in about two thirds.

Diagnosis.—If there be present a tumor, and parasites are discharged with the urine, the size of the tumor diminishing, there can be no doubt. Microscopic examination will determine the character of the milky fluid, or shreds passed. If no tumor can be detected, the discharge of vesicles with the symptoms of nephritic colic will indicate the probable seat of the mother-sac. If a tumor exist without the discharge, it can not be distinguished from hydronephrosis.

Treatment.—The pain and disturbance caused by the passage of the cysts must be treated as renal colic. If the cysts are enlarging, an attempt should be made to destroy them. Electrolysis has been proposed for this purpose, but it appears the attempts which have been made have not succeeded. Injection with tincture of iodine, or with bile, which seems very poisonous to these parasites, should be practiced.

MOVABLE KIDNEY.

Definition.—By this term is meant a kidney become abnormally movable. It is sometimes called *floating kidney.*

Causes.—The kidney may have an unusual mobility, by reason of anatomical peculiarities. The peritoneum may be reflected in front and behind, constituting a *mesos* permitting free motion to the organ. In its natural position imbedded in fat, having the peritoneum in front, and unsupported by ligaments, it is so placed as to be readily dislocated. Should the fat be absorbed, or the peritoneum relaxed, the kidney becomes abnormally movable. This disability is more common in women than in men (ten to two, about), a difference due to two factors—to tight lacing, and to pregnancy. Pregnancy by the great distention of the abdomen, relaxes the peritoneum, and thus removes the principal support. Tight lacing forces the liver down, which pushes the kidney before it, but on the left side the organs have more room. The right kidney is affected in the majority—in Roberts's collection of sixty-five cases, the right kidney was movable in forty-two, the left in nine, and both in fourteen. If the weight of the organ is increased by any cause, the tendency to displacement is proportionately increased. Usually, however, an enlarging kidney contracts inflammatory adhesions to neighboring parts, and thus dislocation is prevented.

Pathological Anatomy.—The congenital movable kidney is distinguished from the acquired by abnormal arrangement of the vessels or peritoneum, or of both. In the acquired mobility the organ is rather elongated, without fat, and detached from the peritoneum. The degree of mobility varies, but the extreme length is not greater than the length of the vessels which form the pedicle. Attacks of perinephritis are common, and hence the kidney may be surrounded by old exudations and bands of adhesion. The dislocated kidney may become attached again and cease to give any more trouble.

Symptoms.—When displaced, the kidney may descend to the margin of the iliac region, but it is usually felt about midway between the inferior border of the ribs and the umbilicus. If the patient is thin, the outline of the organ can be distinctly made out, and it may even be grasped by the thumb and fingers, the pressure producing a sickening pain and faintness. The kidney may also be pushed about, and upward and backward into its proper position, but it will not remain, descending as soon as the support is withdrawn. Respiration changes its position also: it descends on full inspiration; ascends on full expiration. Percussion does not afford a flat note, but a dull tympanitic note. Over the normal site occupied by the kidney, there will be, instead of a flat note on percussion, a hollow tympanitic sound.

Besides the presence of a movable body in the abdomen, which always excites apprehension, there may be no other symptom. In other cases there may be only soreness in the kidney, and a deep-seated sense of aching and pain, with a dragging feeling in the back and loins. Usually, the most pronounced symptoms are those connected with the digestive organs: the appetite is poor, the bowels are constipated, there is much flatulence, and at the same time they suffer from pain in the rambling kidney, and aching and dragging in the loins. This group of symptoms has a paroxysmal character—there are intervals not of entire exemption, but of relief. The intestinal disorders sometimes take the character of cholera morbus, the attacks occurring every few days or weeks, and between them the digestion is troubled, and there is much flatulent distention. Now and then there are cerebral attacks—extreme vertigo, headache, nausea, and vomiting, due probably to twisting of the ureter and retention of urine, congestion of the kidney, etc., and followed by bloody urine, purulent sediment, and finally a copious urinary discharge, the symptoms subsiding. Again, in other cases, there will be much pain and tenderness experienced about the kidney, and requiring confinement to bed, feverishness, a coated tongue, headache, scanty, acid urine, etc.—symptoms probably due to attacks of local peritonitis or adhesive inflammation. In a case of displaced right kidney in a male, there were obstinate constipation, small, flattened fæces, persistent flatus with the sensation of passing an obstacle, due to the position of the kidney against the ascending colon. In all cases, causing symptoms, there is much hypochondriasis, or depression of spirits, even suicidal feelings.

Course, Duration, and Termination.—The cases continue indefinitely. It sometimes happens that the kidney secures firm attachments again, but the author has seen but a single example of such termination. A dislocated kidney is more liable to degenerative changes than a fixed one.

Diagnosis.—As no other tumor behaves as the movable kidney, the diagnosis ought to be easy. The diagnosis rests on these data: the tumor has the shape and size of the kidney; it descends from the position occupied by the kidney, and can be pushed back into the same; it has a special sensibility; the position which the kidney normally occupies is found to be vacant.

Treatment.—As the chief distress arises from the movable condition of the kidney, an attempt should be made to confine it to its proper place by a suitable bandage. The patient must be recumbent, the muscles of the abdomen relaxed; then the kidney is pushed back, a compress is so placed as to prevent its descending, and a closely fitting bandage must then be fastened around the abdomen, so arranged that the support is from below upward. Attention must be paid to the diet, and flatulent-forming food given up entirely. Constipation must be avoided, and the bowels kept in a soluble state. If anæmia exist, a course of chalybeate tonics will be necessary. The secretion of urine should be closely observed, to discover changes in time.

PERINEPHRITIS.

Definition.—By *perinephritis* is meant an inflammation of the loose connective tissue about the kidney. This term is comparable to perityphlitis. As the ordinary result is suppuration, it may be comprehended in the term *perinephric abscess*, as employed by Trousseau.

Causes.—Penetrating wounds, contusions, and even strain (Trousseau) will excite inflammation of the perinephritic connective tissue. Pelvic cellulitis may extend upward by the subperitoneal connective tissue, and ultimately involve the renal. This, although often a puerperal process, may arise from operations on the pelvic organs, etc. Operations on the rectum and inflammatory affections about the bladder may also produce the same result. Chronic pyelitis may extend to and involve the perinephric connective tissue. This disease occurs at adult life till old age, and is more common in men than in women.

Pathological Anatomy.—The connective tissue is at first the seat of an intense hyperæmia ; suppuration soon follows, the purulent elements being mixed with blood, and presenting therefore a grumous aspect; the area of suppuration is not limited, the boundaries of the pus being shreds of breaking-down tissue, the abscess enlarging irregularly. The pus presently becomes yellowish and homogeneous, and something like well-defined limits surround it, but the tendency is to spread along the retroperitoneal connective tissue. An enormous accumulation may take place. The disposition of the abscess occurs in various ways : it may rupture into the peritoneum, exciting general peritonitis ; it may dissect through and discharge externally in the lumbar region ; it may open the colon and discharge by the bowel ; it may burrow along the psoas muscle and open underneath Poupart's ligament, or at the lesser trochanter, etc.

Symptoms.—Pain is a very usual and persistent symptom. Often it begins with the blow or strain, and is a deep-seated aching in the lumbar region, increased by firm pressure, by bending the body, and is not relieved by changes of position, but it sometimes ceases for days, even weeks, but when it returns is more severe than before. With the first pain there is more or less chilliness, followed by fever, general *malaise*, nausea, anorexia, a coated tongue, etc., the fever rising to 103°, 104°, or even higher. The fever has the remittent type, with a morning remission, and there is considerable sweating, especially toward morning. A severe rigor announces suppuration, and chills occur subsequently irregularly, and are followed by high fever and profuse sweats. The body emaciates ; the appetite is gone ; there is vomiting ; an obstinate constipation, requiring active purgatives to relieve it, comes on ; the skin acquires the yellowish, earthy hue or fawn-color of suppuration. After a time, a swelling is discovered in the flank, and the depression, which normally exists in the lumbar region, assumes a convex shape. On careful manipulation, deep-seated fluctuation may be detected. If left to pursue its course undisturbed, the pus finally points in the lumbar region. The pus may be odorless, or it may have a fecal odor without any communication with the bowel. If the abscess discharges, and there is no complication, the condition of the patient at once improves, the fever ceases, the appetite returns. If the pus burrows downward, the duration is more protracted, and there is much pain, the abscesses opening in the groin. Discharge taking place by the lumbar region, extensive emphysema, occupying the whole extent of the back, may occur (Trousseau). In such cases communication is established with the bowel, and hence the emphysema is due to the intestinal gases. Fæces may be discharged by the lumbar opening, and recovery ensue. If rupture into the peritoneal cavity occurs, intense peritonitis, with the usual symptoms, will be excited. Rupture into the pelvis of the kidney will be announced by the sudden discharge of pus in the urine.

Course, Duration, and Termination.—The symptoms are very obscure until the fluctuating tumor appears ; the cases then pursue a very uniform course, and the primary form, rapid course. Discharge of pus may terminate an uncomplicated case in three or four weeks. Recovery is the usual termination in such cases. Extensive and protracted suppuration will induce a typhoid state and death by exhaustion. Rupture into the intestinal canal is rapidly fatal. When communication is established with the colon, recovery may ensue, but the result is doubtful. When the abscess is secondary to puerperal processes, the termination is usually in death. In a few cases, the inflammation of the perinephric tissue undergoes resolution without suppuration. The morbid process may produce or succeed to pyelitis, or the kidney itself may become diseased—results which aggravate the existing disease.

Diagnosis.—Perinephritis may be confounded with hydronephrosis, echinococcus, and cancer. In all of these diseases a tumor exists : in perinephritis, accompanied by fever and sweats and the other evidences of suppuration ; in hydronephrosis and echinococcus, an enlarging tumor without pain ; in cancer, a painful tumor and hæmaturia. Perinephritic abscess tends outwardly to point in the lumbar region, or downward, in the groin, while the other tumors grow forward and downward into the peritoneal cavity. Pyelitis with tumor is distinguished from perinephritis by the condition of the urine.

Treatment.—With the first symptoms, leeches may be applied to the lumbar region, followed by ice. Purgatives should be administered. If there is much pain, morphine is necessary. Large doses of quinine (ten grains every four hours) should be given with the view to check the migration of the white corpuscles, and preferably with morphine, although the pain may not be great. As soon as suppuration occurs, supporting measures are required. Malt liquors, a generous diet, alcoholic liquors, and quinine are the most appropriate means. A free incision should be practiced as early as possible, and drainage established.

SOME DISEASES OF THE BLADDER AND URETHRA IN THE MALE.

CLINICAL EXAMINATION.

[Note.—Diseases of the bladder and urethra are usually considered surgical maladies, and do not appear in a medical treatise; but as they so often come into relations purely medical, and demand the kind of consideration which a practical physician gives to all his work, I have decided to incorporate some account of them in this edition].

I have already given (page 503) an outline of the modes in which urine is studied for clinical purposes. It is necessary to add, however, some special points in respect to vesical troubles.

When disease of the bladder is supposed to exist, the urine of twenty-four hours should be collected, and its appearance compared with known specimens of the normal fluid.

There being simultaneous disease of the urethra, a drop of the urethral discharge should be compared with a drop of the urinary sediment coming from the bladder. In this way valuable information is got as to the source of a given specimen of urine in disease of the urinary passages.

In catarrh of the bladder, whether acute or chronic, the action of extraneous causes must be taken into account. A gonorrhœa, recent or that has suddenly ceased to flow, some obstructive difficulty as an urethral stricture, enlarged prostate, etc., or some reflex irritation, as hæmorrhoids, etc., may be the source of the mucous inflammation, rather than any intrinsic trouble.

It is necessary, therefore, to be provided with means for investigating the condition of the urethra. For this purpose, bulbous sounds* are necessary if the lesion is a stricture. These were at first graduated according to the French scale, and extended from No 1 (·03 in diameter) to No. 40 (in diameter, 13·33). At present, chiefly through the remarkable work and practical demonstrations of Dr. Otis,* we have an American scale, the sounds graduated in the metric system, and increasing *one millimetre in circumference*, up to No. 40, the maximum size.

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* From Van Buren and Keyes's "Treatise." Published by D. Appleton & Co.

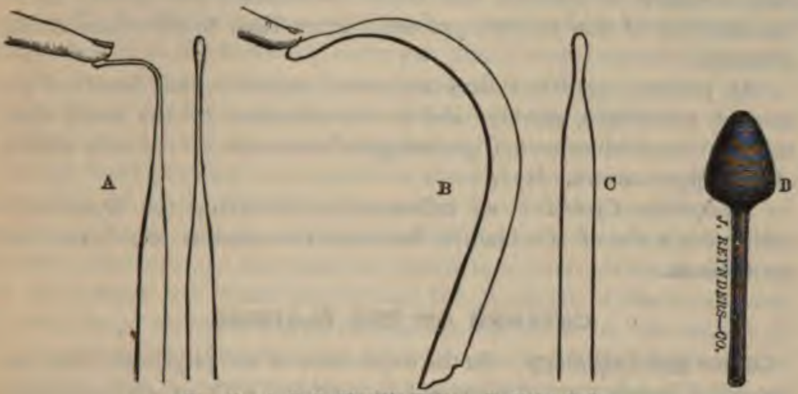

FIG. 45.—A, B, C, Flexible Bulbs. D, Metal Bulb.

To determine the normal size of any urethra, it is necessary to make use of a measuring instrument, graduated in the same scale. This introduced and gradually expanded up to the proper point will necessarily give an approximation to the proper measure of the canal, but it must be remembered that this tissue is extensile, and too great force will furnish an abnormal result.

If the canal be narrowed by any thickness of deposit, a catarrh of the bladder will be produced ultimately. Hence it follows that the stricture must be removed.

Enlarged prostate so often figures as a cause of vesical catarrh that grievous errors are sometimes committed—errors of opinion and errors in the treatment. The age of the subject must be taken into account. Enlarged prostate may exist without changing the caliber of the urethra, it has been proved, and much obstruction may be due to changes in the isthmus of the prostate so situated as to narrow the caliber of the canal at the very entrance of the bladder, and yet not cause any demonstrable change in the body (in the two lobes) of the gland.

No mistake is more common than confounding stricture with the triangular ligament, through which the urethra passes, and against which it doubles up when a sound or catheter is hastily and indiscreetly passed.

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* "Practical Clinical Lessons in Syphilis and Genito-Urinary Diseases," New York, 1883, p. 437.

FORMS OF CYSTITIS.

The term *cystitis* means an inflammation of the bladder, which may be *acute* or *chronic*, but these distinctions are merely as to the severity of the morbid process, rather than to the time it has occupied.

At present, cystitis means an acute catarrh which involves the mucous membrane, merely, and is characterized by the usual sign, viz., an overproduction and pathological alteration of the cells chiefly of the upper or outer layer.

Interstitial Cystitis is an inflammation involving the layers composing the walls of the bladder between the mucous membrane and peritoneum.

CATARRH OF THE BLADDER.

Causes and Pathology.—In the experience of the physician there are two chief causes: an ill-managed gonorrhœa, with or without stricture; disease—rarely tuberculosis—especially obstructive conditions affecting the isthmus or body of the prostate gland. It sometimes happens that suppurative pyelitis extends downward, and ultimately the bladder takes on the catarrhal process. There is reason to believe that persistent excess of uric acid may also start an irritation that ultimately becomes catarrh of the bladder. The agency of calculi and other foreign bodies are well-known causes in the experience of surgeons.

More frequently than uric acid, does the phosphatic condition determine attacks of vesical catarrh. It must be understood, however, that the production of alkaline urine is usually the result of pre-existing disease of the urinary passages, especially of retention of urine and decomposition, whereby ammonia is formed from the urea. The alkaline state of the urine changes the appearance and condition of the mucus, which becomes semitransparent, tough, glutinous, and "stringy."

Symptoms.—Catarrh of the bladder, when secondary to a chronic urethritis, may come on insensibly to the patient. There is at the outset little more than increased irritability of the bladder—frequency of discharge—but of the same character as before. Soon, however, especially if the case takes on more of an acute character, the vesical irritability is accompanied by a deep-seated soreness felt in the perineum, rectum, and along the course of the ureters. The calls to urinate become more frequent and urgent, the flow is accompanied by straining, burning pain, and only a few drops may slowly distil out, as it were, or only muco-pus is forced out, it may be, and this streaked with blood. The sense of relief experienced when the bladder is emptied, becomes shorter in duration, and presently is no longer felt, the vesical tenesmus continuing present all the time. At length rectal tenesmus comes on, hæmorrhoids—large, sensitive, and bleeding—may form and contribute materially to the sufferings of the patient. The urine is acid or alkaline, the latter in cases of long duration, is voided in small quantity at a time—it may be drop by drop—with extreme anguish, and so thickened by muco-pus that it flows imperfectly.

If the enlarged prostate is the cause of the disease, to the symptoms of the bladder catarrh are added those pertaining to the enlarging gland—a deep-seated soreness with a sensation of a large foreign body pressing for expulsion through the rectum. If gonorrhœa has set up the catarrh, the onset of the bladder trouble may be silent, and not until an excessive irritability comes on is the complication observed. In the case of obstruction from stricture, the size of the passage left enters largely into the character of the symptoms. Thus, a large stricture which encroaches but little on the caliber of the canal will be accompanied by a gleety discharge which proves obstinate, and in consequence of the attention given to this, the occurrence of a catarrh of the bladder may be unobserved, until an unwonted frequency and slowness of the urinary discharge demands treatment. When the stricture is a tight one, the increased embarrassment attending the flow of urine at once is recognized by the patient.

From the beginning of a vesical catarrh the discharge of urine becomes troubled, but when serious obstruction exists in the urethra, such an active catarrhal process is set up that soon the flow is started with difficulty; but a part of the contents of the bladder is discharged, and in consequence the residual urine constantly increases, decomposition goes on, the contents of the organ become highly alkaline and offensive, and the muco-pus is produced in enormous quantity. The mucous membrane, also, excretes phosphate of lime in such a large amount that it forms with the muco-pus a thick adhesive coating which has become an immovable lining of the whole interior.

When it has attained advanced proportions, catarrh of the bladder affects the general state to a greater or less extent. A septicæmic fever may occur. The paroxysms have a characteristic irregularity in the time of occurrence, but in the manner of acting bear a close resemblance to intermittent fever. A severe chill, followed by fever (temperature, 102° to 105° Fahr.), and a profuse sweat, which is apt to be prolonged, even to twelve hours or more, are the symptoms that constitute the complexus of the septicæmic fever. The frequency with which these paroxysms occur depends on the severity of the catarrh, and on accidental circumstances. Malarial infection, acute indigestion, irregularities of conduct, and exposure to cold and damp, are among the accidents that most frequently cause a recurrence of the paroxysms. The close resemblance of the septicæmic to the malarial fever is shown in the behavior to the action of certain remedies, as well as in symptoms—in the reactions under quinine especially.

Course, Duration, and Termination.—Catarrh of the bladder manifests no disposition to spontaneous cure. If permitted to continue undisturbed, the catarrhal inflammation increases in area and depth, and spreads widely by contiguity of tissue. The most serious complication that ensues by reason of contiguity is an extension to the kidneys, the development of pyelitis, and subsequently of multiple abscesses. When catarrh of the bladder is produced by obstruction, obviously its subsequent behavior is determined by the changes in the obstructing causes. A stricture may be cured, and when this is accomplished, the catarrh can be managed easily and effectively, but when chronic enlargement of the prostate is the source of mischief, only amelioration can be expected ; but that amelioration has a high degree of value, and is only short of cure in the comfort wrought.

In the study of catarrh of the bladder, the actual state of the urethra, or prostate, must be ascertained. Although, properly speaking, a surgical procedure, every medical practitioner should be prepared to sound the urethra and bladder. Three instruments only are essential : bulbous sounds of various sizes, a curved metal sound, flexible catheters of vulcanized India-rubber or caoutchouc, and prostatic silver catheter. The bulb-sound is indispensable for ascertaining the situation and caliber of a stricture. Rendered aseptic, and lubricated with prepared vaseline, the sound first tried having the size of the healthy urethra, is passed gently down the canal ; when a narrowed part is reached the fact is recognized by the resistance felt in passing through, but especially by the resistance and sudden jerk combined, felt when the sound is drawn back through the stricture. If stone is present, the silver prostatic catheter will probably disclose it.

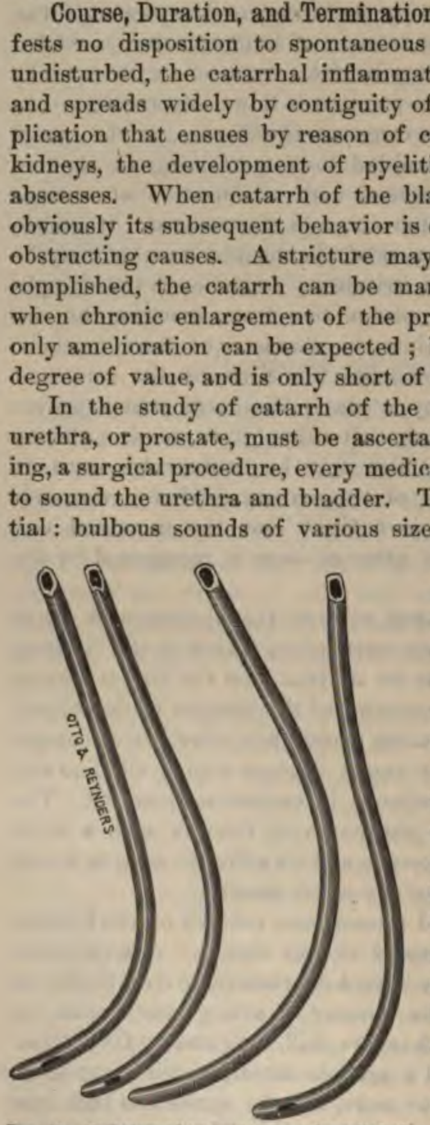

FIG. 46.—Catheters of various curves, for relief of bladder.

The course of treatment in various cases has much to do with the result. Appropriate local and systemic management is largely efficacious in certain kinds of cases. If the disease pursues its own way unaffected by treatment, various secondary troubles arise. Extension by contiguity of tissue takes place along the ureters, and thus serious changes are slowly set up in the kidneys.

In course of time, extension of the malady takes place through the other layers of the bladder, and serious pathological changes occur.

Treatment.—It must be admitted here that the methods of treatment pursued by the surgeons are more efficient than those employed by physicians; but it will be found, I think, that a combination of the two is better. Remedies that can effect the vesical mucous membrane only by passing through the system, are shorn of much of their power by diffusion, yet they accomplish undeniable good.

There are numerous remedies of merely empirical origin that have been popular, but it may well be doubted whether the good is not surpassed by the injuries inflicted on the mucous membrane by their irritating contact. Copaiba, cubeb, sandal-wood oil, and others of the same character, are the most evil of the group, and I advise my readers to prefer more enlightened methods.

Of the remedies acting on catarrh of the bladder, in acute cases, the most useful are the alkaline bicarbonates of sodium or potassium, especially of potash. The ordinary neutral mixture effervescing draughts in which bicarbonate of potassa is given in excess, lemonade made of bitartrate (cremor tartar), some lemon juice added and sweetened to the taste, are all useful by diluting the urine and lessening acidity. When the urine is alkaline, and contains much lime phosphate, the alkaline salts are hurtful, and mineral acids become appropriate remedies. When the catarrhal process is at a more advanced stage, the kind of remedy given depends on the state of the urine. If acid still, to the remedies above mentioned may be added tincture of cantharides (5 to 20 m.), and oil of erigeron (same dose), remedies that are really effective when the acuter symptoms have subsided. The exacerbations of fever that occur in the course of the chronic form of vesical catarrh are most successfully treated by antipyretic doses of quinine. They are not unfrequently mixed with malarial toxæmia, when the rule of the recurrent paroxysms is regularly intermittent. These febrile attacks are septicæmic in kind, and are due to the suppuration occurring in the bladder. Only massive doses (15 to 20 grs.) will be effective under these circumstances. Antipyrin, acetanilid, resorcin, and other members of the antiseptic group, are also used with, or as substitutes for the quinine. I am of opinion that quinine is more effective, really, than any of them, if firmly but judiciously administered. When suppurative pyelitis becomes an element in the morbid complexus, no curative result should be looked for.

As stricture of the urethra is an important factor in the causation of the vesical catarrh, so its removal comes to be an essential element in any curative process proposed. As now the most prompt and efficient means of treatment consists in electrolysis, physicians should have a proper equipment for such purposes, a competent knowledge of the science of galvanism, and a practical acquaintance with all the details necessary in the electrolytic treatment of stricture. Without these accomplishments the operator will either fail miserably, doing more harm than good, or he will succeed by accident merely, and not know in what his success consists. The following appliances are necessary:* A galvanic battery that will furnish five to fifteen milliampères of current strength, as shown by the milliampèremeter; insulated urethral electrodes, with button or cylinder of a shape to enter the stricture or to lie in contact with it. No pain should be caused by the application, and at the outset the current should be quite mild. The *negative pole* is connected with the urethral electrode, and the current is allowed to flow uninterruptedly from five to twenty minutes.

When the stricture will admit a sound of full size, the so-called "gleet," which is always present in these cases, will cease. The canal sufficiently dilated, the catarrh of the bladder can then be successfully treated.

ACUTE URETHRITIS—GONORRHŒA, OR SPECIFIC URETHRITIS.

Definition.—Gonorrhœa is an acute catarrh of the urethra caused by the development or *pullulation* of a parasite—a *gonococcus*. The purulent discharge containing this organism will excite the same kind of action on other mucous surfaces of the same individual or of other persons. It is therefore a contagious malady, and is most usually transferred by the sexual act.

Symptoms and Pathology.—In what mode soever the poison may be transferred the morbid process begins at the point of contact—the prepuce and entrance of the urethra. After a variable period the local action becomes more intense—a sensation of heat and irritation calls attention to the part, and then an area of redness and swelling about the meatus and neighboring mucous surface of the prepuce is seen covered with a creamy pus. A little pressure will force out of the meatus a drop or more of light purulent matter, and a feeling of soreness is developed. From the time of suspicious contact up to the actual onset of gonorrhœa is a period which varies greatly, but, it may be said, is rarely less than two days nor more than eight days; but authentic examples of much longer periods are quite numerous.

The local action increases rapidly; the whole under surface of the prepuce becomes deeply engorged, much swollen, and covered with a creamy pus. Attempts to retract causes great pain and can not usually be done. When the foreskin is elongated and covers the glans penis, and covers the glans in the normal state, accumulation of pus takes place beneath, the small orifice may be so obstructed that the urine is passed with difficulty, and great pain is felt even when an inspection of the meatus is attempted. The perineum and deeper parts become the seat of a heavy tensive and sore feeling, the bladder becomes more irritable, and when urination is attempted a condition of strangury comes on. The testes are somewhat sore and tender, and more or less pain is felt along the spermatic cord. At night all the distressing sensations are intensified; erections occur in which the penis is half-filled, and bent with a sensation of desire to urinate, every movement only increasing the distress, and if any water is passed it is drop by drop with extreme anguish. These half erections are known by the French term "chordee." Nocturnal seminal losses, also, occur, but with painful erections, and increased distress follows. The inflammation and swelling may extend to the prostatic urethra and involve the *sphincter vesicæ*, causing retention of urine, or extreme irritability and frequent voiding of a few drops of bloody urine with great straining and severe pains extending through the pelvic cavity into the hips, perineum, and down the thighs. Meanwhile a copious discharge of yellow pus, often of ichorous pus, continues from the urethral orifices.

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* The reader will find in my treatise on "Medical Electricity," third edition (Lea Brothers & Co., Philadelphia, 1886), a full account of the mode in which the treatment is carried on.

Such is a mere outline of the symptoms of a fully developed attack. It is less important, however, to enter into minute details regarding the symptomatology than to set forth the complications which arise in its course and the sequelæ that give origin to numerous troubles requiring the aid of the physician.

Course and Duration.—So much depends on the kind of treatment pursued, and on various personal and social influences to which the patient is subjected, that no fixed rules as to the course of any case can be laid down. If the patient will remain in bed, have a saline laxative and a spare diet, the case will not get nearly so severe, and will subside early and disappear in from two to three weeks.

The complications that belong to gonorrhœa, have a high degree of practical importance. During the acute attack, an extension of the morbid action to the testes is one of the most painful and serious of the lesions, for closing of the spermatic ducts may ensue, and this means inability to procreate ever after. Gonorrhœal rheumatism may supervene, and this is slow in getting well. Stricture of the urethra may be a result of the disease and injudicious treatment combined. Catarrh of the bladder may be developed by extension of the inflammation by contiguity of tissue.

Treatment.—There is no disease in which skill in treatment has more influence over the result than gonorrhœa. The curative result is much facilitated by quiet, but as in most subjects attention to business is necessary, the disease is rendered more severe and more difficult to treat. A saline laxative should be given, a suspensory bandage put on, and the parts shielded from injury. As a gonococcus is the active agent in setting up the inflammation, the treatment should be parasiticide, and as a local affection it must have local application.

The first step consists in washing out the canal to and a little beyond the point to which the disease extends with hot water—as hot as can be borne. Before an injection is practiced the patient should pass water to clear out the canal as far as possible. A small fountain syringe and a double catheter-tube with inlet and outlet form the best mode of washing out and applying medicaments to the canal of the urethra. Adhesive materials have a special advantage in that they remain long in contact and have consequently a much greater effect. Such bland remedies as bismuth, especially the salicylates, in an emulsion of gum makes a highful effective combination for topical application. As the salicylate is not a sufficiently powerful parasiticide, for this purpose the corrosive chloride or the biniodide of mercury may be added to the injection in minute quantity. Late researches have shown that the $\frac{1}{1000}$—even the $\frac{1}{2000}$—has proved effective against the pathogenic micro-organisms and their spores. This corrosive chloride in the smallest effective quantity should be added to the injections. The inexperienced practitioner must be warned against the use of strong injections. Nitrate-of-silver solutions have done much harm, especially when the so-called abortive injections were in use. It is now the practice to use weak injections—solutions of mineral salts, lead, copper, zinc—and frequently. It is probable that the result is due to the mechanical displacement of the mucus containing the gonococcus, rather than to any direct action on the mucous membrane.

To accomplish the best results in acute gonorrhœa, the following, or, some similar combination may be used: ℞ Plumbi nitrat. *vel* bismuthi salicylat., ʒ ij; hydrarg. chlor. cor., gr. ss.; mucil. acaciæ, aquæ, āā ℥ ij. M. S. Use one or two teaspoonfuls at an injection. In this combination the proportion of corrosive sublimate is about $\frac{1}{1000}$.

Carbonate of lead, subnitrate or subcarb. of bismuth, are bland preparations, which may be used with a certainty of good results. It is important in making an injection that sufficient mucilage be added to give it such adhesiveness that it will continue long in contact with the diseased surface—until, at least, the next injection is given. The patient should also clear out the canal by passing water, if he can, before taking the injection, and also use a douche of hot water in advance.

URETHRAL FEVER, OR CATHETER FEVER.

Some of the complications to which reference has been made are more especially surgical, but the author has some observation to make on the treatment of urethral fever, inflamed testes, and stricture, from the point of view of medical practice.

Urethral fever is a name given to a febrile paroxysm having the septicæmic character—a chill, fever, and sweat—but the duration of the seizures and their periods of recurrence are by no means regular. Such an attack may be brought on by passing a catheter, and is then called catheter fever. The passage of a sound, irrigation of the bladder, even simple operations on the outer parts of the canal, will be promptly followed by an attack in some subjects. The character of the subject has much to do with the occurrence of the seizures—for the nervous, those with an irritable nervous system, are specially susceptible, whereas the lymphatic are usually but little disturbed by these operations. It must be understood, however, that a urethra through which an instrument can be passed without evoking any sensation is not normal, and is a bad indication in cases of functional impotence.

When such a susceptible state exists, a comparatively short period supervenes before the chill begins—in some instances, at once. The chill is really a severe rigor sometimes, and profound depression ensues. Indeed, collapse ending in death has been the result in a few instances, it is true, but often enough to demand caution. Care is necessary in all cases even when there is the least probability of dangerous symptoms. A full dose of quinine (15 gr.) and of morphine (¼ gr. to ½ gr.) will usually prevent trouble or diminish the danger of hyperpyrexia ensuing, or, at least, lessen the extent and persistence of febrile heat. Next to this must be mentioned the recent antipyretics, such as antipyrin, acetanilid, and the salicylates.

INFLAMMATION OF THE TESTES.—If the gonorrhœal inflammation extends to the testes, the case is made much more formidable. Few cases of urethritis are without soreness and tenderness of the testes. This fact evident, care should be exercised to prevent the smallest injury to the organ. The cases in my experience almost always occur in boys and young men who have gonorrhœa for the first time. They try to escape detection, and play and expose themselves as if nothing were the matter. The consequence is they either bruise the organs or practice such strong injections in the hope of speedy relief, that the swelling is soon brought on.

When the inflammation begins, and before decided swelling ensues, there is felt in the lower abdomen a colic-like sensation which may mislead an inexperienced practitioner; but on closer inspection it will be found that the real site of the pain is along the course of the spermatic cord to the bladder. The actual inflammation followed by swelling usually occurs in the epididymis, which is found to be hot and very tender. This may be the sole seat of inflammation, or it may precede by several hours or days the inflammation of the body of the organ.

It should be understood that inflammation of the epididymis has a high degree of importance from the point of view of the function of procreation. It is now known that if both organs (epididymis) are so much inflamed that the spermatic duct is closed, no semen can pass from the testes, and hence if both are blocked, impotence results. The power of erection is not impaired at once, but after a time intercourse becomes less and less complete, and is extinguished long before the time usual in the normal condition of things. When the inflammation reaches its highest point the pain is great, and any attempt to move the organs causes exquisite suffering. When the inflammation reaches the highest point, it remains nearly stationary for several days. The gonorrhœal discharge, or the gleet, usually ceases when the inflammation begins, and to its disappearance the patient attributes the development of the trouble in the testes. Professional opinion formerly acquiesced in this view, and to a limited extent this may be admitted; but it is to the migration of the gonococcus and the development of colonies of micrococci that the extension of disease is due.

DISEASES OF THE NERVOUS SYSTEM.

CLINICAL EXAMINATION — MODES OF ASCERTAINING THE STATE OF THE NERVOUS APPARATUS.

Cerebrum.—The examination into the functions of the cerebrum includes the study of the mental condition, of the organs of special sense, and of the state of common sensibility in the area of distribution of the sensory nerves supplying the head and face.

The *intra-cranial circulation* is investigated through the facial vein, and the appearance of the membranum tympani and the retina. When an obstacle to the intra-cranial circulation exists sufficient to compress the cavernous sinus, the conjunctiva is injected, the eyelids swollen, and the nasal mucous membrane is congested and bleeds readily. These results come from the anatomical connection between the facial vein and the pterygoid plexus of veins. When there is cerebral congestion, or anæmia, the membranum tympani exhibits a more or less vivid redness, or an appearance of pallor.

Ophthalmoscopy.—The retinal circulation being a diverticulum of the cerebral, valuable information is gained by ophthalmoscopic examination. The ophthalmoscope used for this purpose should be a metal concave mirror, of ten or twelve inches focus, with a revolving disk behind it provided with ocular glasses. Loring's or Knapp's are well suited to this purpose. The observer should be provided also with two convex object glasses, having two to four inches focus, and a concave lens—the latter for the direct method of examination.

Both the *direct* and *indirect* methods of examination are to be employed, as a rule. In the former the eye-ground is illuminated by the mirror, and the observer, seated close to the patient, looks through the pupil down on the retina, as one would look into a closed room through the key-hole of the door (upright image). In the indirect method (inverted image) the light is thrown into the eye as before, but a double-convex lens, held between the thumb and index-finger, is interposed in front of the eye under examination, the hand being supported by the little finger resting on the patient's forehead. In this way the focus can be readily adjusted.

The simplest appliances suffice for such ophthalmoscopic examination as may be required in ordinary clinical work. A small kerosene-oil lamp will furnish the light, or, in the absence of this, a candle even may be utilized for the purpose. Although such an examination will not be sufficient for any important scientific purpose, it will afford more or less valuable insight into the condition of the intracranial organs, and suggest the course for future and more accurate investigation.

The changes occurring in the retina or the "eye-ground" will be mentioned hereafter in connection with the maladies of the brain.

Impairment of vision, amblyopia, amaurosis, hemiopia, diplopia, etc., alterations of the accommodation, deviations of the ocular globe from paralysis or spasm of the eye-muscles, the size and sensitiveness of the pupil, may be symptomatic of intracranial disease, and will be discussed in their proper relations.

Otological examinations are necessary in all cases of cerebral disease, whether or not the ear appears to be directly affected. It has already been stated that the condition of the intracranial circulation may be ascertained by an inspection of the *membranum tympani.*

The hearing power can be measured by the ticking of a watch, by the tuning-fork, and by the voice. The distance from the ear the tick of a watch can be heard by the normal individual, is the standard with which the hearing power is to be compared. For example, if the watch-tick is audible by the normal ear at a distance of six feet, and by the diseased ear at one foot, the hearing power would be stated as $= \frac{1}{6}$. The voice is a more accurate measure, and the hearing should be tested by distinct tones, and by whispering at a specified distance. The tuning-fork (of the note C) is used more especially to determine the condition of the auditory nerve, and is placed in contact with the incisor teeth or forehead. If the patient is deaf to the watch-tick and voice, he may still hear the tuning-fork, showing that the difficulty is in the sound-conducting apparatus, and not in the nerve, which yet transmits the sound vibrations.

The apparatus required for the investigation of the auditory complications in cerebral diseases is, besides those mentioned above, a suitable ear speculum, and a concave mirror with a central hole, and attached to a convenient handle with a universal joint.

The condition of the hearing power comes into relation to various intracranial diseases, such as tumor, abscess, meningitis, etc. Lesions of certain parts of the ear cause symptoms of cerebral disease—for examples, Menière's disease or labyrinthine vertigo, etc.

The electrical reactions of the auditory nerve as determined by the polar method have some diagnostic value.

The *sense of taste* may be studied by placing sapid substances on the tongue, and noting the time when their character is appreciated, or the diminution in acuteness or the entire absence of the sense. The tongue should be well protruded, and the eyes closed. Then, by means of a small spatula, or brush, the substance to be tested is placed on that part of the tongue supposed to be affected, and is allowed to remain until contact with the nerves is assured. Salt, sugar, quinine, and vinegar may be used to determine the appreciation of the saline, sweet, bitter, and acid sensations respectively.

For the electrical reactions, see *post.*

Spinal Cord and Nerves.—The examination of this part of the nervous system includes *sensibility*, *motility*, and the *reflexes.*

Sensibility.—In the absence of special contrivances, sensibility may be tested by the simplest means. With a needle-prick, pinching the skin, pulling on hairs, the presence or absence of common sensation can be ascertained, and differences noted by comparing symmetrical parts. The faradic current is the best mode of ascertaining the ab-

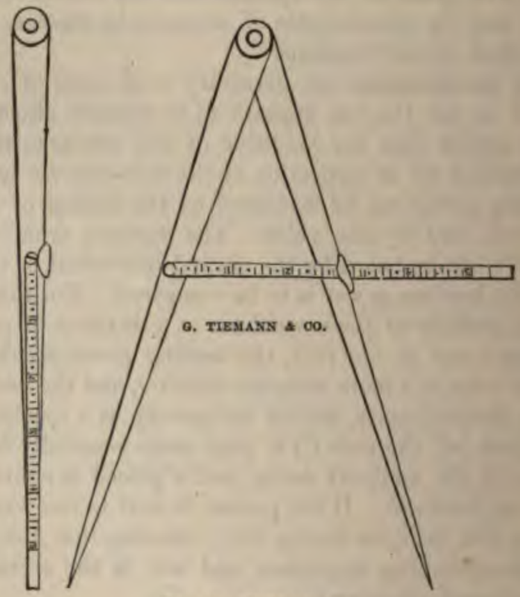

FIG. 47.—Æsthesiometer.

sence of the sense of pain (*analgesia*). In testing in this way, the skin is carefully dried, and some drying powder (infant powder will answer) is dusted over the surface to remove all moisture, so that the current may be limited to the skin. A metallic electrode of small size is then passed over the affected area and the neighboring normal integument. The limits of lessened or absent sensibility to pain are, in this way, exactly indicated, and can be marked out.

The tactile sense is best studied by means of the *æsthesiometer*, an instrument originally devised by Sieveking.* The most convenient form of this instrument is that suggested by Hammond, and shown in Fig. 45. It consists of a pair of dividers, to one arm of which a scale is attached for indicating the distance of the points apart.

Works on physiology contain tables exhibiting the relative acuteness of the tactile sense of various areas. A few of these may be mentioned as a guide. The points of the æsthesiometer can be distinguished as two, at a distance apart—

On the tip of the tongue of	·5	line.
Palmar surface of index-finger	1	"
End of the nose	3	lines.
Palm of the hand	5	"
Back of the hand	8	"
Forehead	10	"
Back of the foot	18	"
Front of the thigh	30	"

Several circumstances affect the results: attention and practice on the part of the patient increase the acuteness. When the points are separated transversely to the direction of the limb, they are more readily perceived than when placed longitudinally; also, when put on one after the other, and when the instrument is moved along the surface.

It is necessary, to secure accuracy, that the patient do not guess; that the points of the instrument be blunted, so that pain may not interfere, and that the temperature of the compass be that of the body, so that the impression of heat or cold may not increase the readiness of perception.

The *sense of temperature* may be ascertained by the application of hot and cold bodies. Test-tubes containing cold and hot water at a known temperature can be put on the affected regions, and the acuteness of perception compared with that of normal parts.

Allochiria is a peculiar state in which the patient is unable to say on which side he has been touched, or refers the sensation to the wrong side.

The *sense of pressure and of weight* is determined by placing weights on the part to be tested, superimposed, so that the least difference can be noted. Eulenburg* has invented for this purpose an instrument called the *baræsthesiometer*, which is a spring having a graduated scale attached, on which are registered varying degrees of pressure. Ordinary brass weights, or bits of metal of definite weight, can be used for this purpose. They must be sufficiently varied to permit a nice discrimination between the sense of the normal and of the diseased part.

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* "The British and Foreign Medico-Chirurgical Review," January, 1858, p. 251.

Rate of Conduction of Sensory Impressions.—In diseases characterized by impaired sensibility, the rate at which impressions move to affect the sensorism is below the normal, and when hyperæsthesia exists it may be above. In some cases the retardation may be sufficient to be recognized by ordinary means, but usually very delicate apparatus is necessary to determine the time. The various modes of sensibility may be interrogated in turn—touch, pain, taste, vision, and hearing.

Motility.—The motor mechanism to be studied embraces the voluntary and the automatic.

The gait of the patient should be carefully observed—in walking, running, standing on leg, etc. The movements of the hands in writing, in touching the tip of the nose with the eyes closed, in buttoning clothing, etc., should be watched, and deviations from the normal noted. Weakness of the hands can be measured by means of the dynamometer (Fig. 46); and by the dynamograph (Fig. 47), tremors, unsteadiness, early fatigue of the muscles, may be represented graphically. In using the dynamometer the spring is grasped in the hand, and the whole strength that can be put forth is measured on the graduated scale of the instrument.

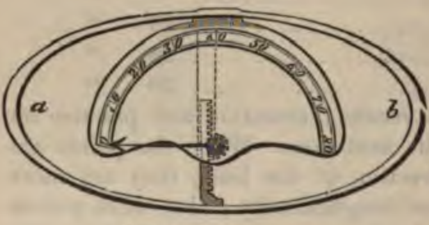

FIG. 48.—Dynamometer.

The automatic movements to be observed are those of the iris, of the respiration, and of walking. The mechanism of locomotion is governed by a voluntary and automatic regulator. The effect of disease in this locomotive apparatus is seen in the character of the gait. For example, the gait of ataxia, the reeling movement in disease of the cerebellum, and the ataxia and reeling combined in affections of the peduncles of the cerebrum.

Electric Excitability.—In determining the state of the voluntary muscles, both faradic and galvanic excitation must be employed. In electrical stimulation we possess the best means of ascertaining the state of the muscles. The applications of the electrodes are *labile* and *stabile*, *direct* and *indirect*—labile, when the electrode is moved over the surface under examination; stabile, when kept in one position; direct, when the electrodes are applied to the muscle to be tested; indirect, when the muscle is stimulated through the motor nerve supplying it.

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* "Lehrbuch der functionellen Nervenkrankheiten," etc., Berlin, p. 17.

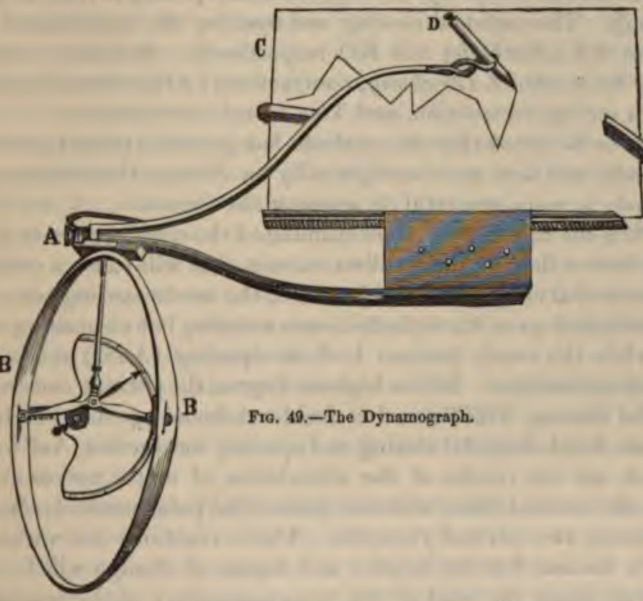

Fig. 49.—The Dynamograph.

When the muscles are healthy, the weakest current that can induce a muscular contraction will have an equal effect whether the motor nerve or the muscle be stimulated. In using the faradic current for determining the state of the voluntary muscles, only the weakest current that will cause contraction should be used. An increase in the readiness of response may be noted; more frequently there ensues a quantitive decline, and it is found that not only are stronger currents required to bring about a contraction, but that with a current of definite strength the contraction produced is feebler than in health.

The excitability to the faradic current may entirely disappear, and no strength of current cause any contraction.

In using the galvanic current, it must be remembered that muscular contractions ensue on opening and closing the circuit. There is a well-defined law of muscular action under the stimulus of the galvanic current; it is called the *normal formula*, and the method by which it has been ascertained is designated the *polar method*. Certain symbols in imitation of the chemical have been agreed on as a means of giving ready and precise expression to the data. The polar method consists in the application of the pole, the reactions to which are to be determined, to the nerve or muscle, while the other rests on any indifferent point, as the sternum, thigh, etc. Closing the current by the application of the anode is anodal closing, and is represented by the symbol AnS (*Schliessung*, closing); anodal opening is AnO (*Oeffnung*, opening). The cathodal closing and opening are represented by the symbols KS (*Kathode*) and KO respectively. Muscular contraction has for its symbol Z (*Zuckung*, contraction); z for a weak contraction, Z′ for a strong contraction, and Te a tetanic contraction.

It is to be noted that the cathode has greater excitant power than the anode, and acts more energetically on closing the circuit, whereas the anode is more powerful on opening the circuit.

When the motor nerves are stimulated the reactions are as follows: In the lowest degree, the weakest current that will cause a contraction is the cathodal closing contraction, KSz, the anode causing none. In the intermediate degree, the cathode causes a closing but no opening contraction, while the anode induces both an opening (AnSz) and a closing (AnOz) contraction. In the highest degree, the current causes tetanic cathodal closing, KSZTe, and a feeble cathodal opening, KOz, while there are decided anodal closing and opening contraction, AnSZ, AnOZ.

Such are the results of the stimulation of motor nerves and muscles in the normal state, with the poles—the polar method—and hence the phrase, the *normal formulæ*. These reactions are variously altered in disease, but the manner and degree of change will be defined hereafter, under the head of the symptomatology of the several affections concerned.

The Reflexes.—To every reflex action the following mechanism is necessary: A point of perception of the impression or irritation; afferent fibers of communication; the center; efferent fibers for transmitting the reaction outwardly. The reflexes may be deranged in two ways: they may be heightened or exaggerated—*reflex hyperkinesis*; they may be wanting—*reflex akinesis*. The cutaneous reflexes are those due to irritation of certain parts of the integument, followed by muscular contractions in areas anatomically associated therewith. They have, consequently, a high degree of significance, and are of great importance as a means of ascertaining the condition of the spinal centers. The deep reflexes consist of muscular contractions, caused by percussion of the muscles or of their tendons. They have even higher significance than the cutaneous reflexes.

The *eye reflexes* to be observed are the closure of the lids on irritation of the conjunctiva, and contraction of the pupil on exposure to the light. The movements of the iris may be studied as follows: Closing one eye, the outspread hand is passed in front of the other eye. The shadow thus caused sensibly affects the healthy iris. The rate and degree of movement, or the absence of movement, should be noted.

The *palmar reflex* consists in contraction of the fingers on tickling the palm, but this can be seen only in infants, or in adults during sleep or unconsciousness, since the brain in activity exerts a controlling inhibitive influence on the spinal reflexes.

The *reflexes* of the scapula, of the erector spinal muscles, of the epigastrium, and of the abdominal muscles, are all produced in the same way—namely, by stimulation of the skin of these regions respectively, and they all depend on a normal condition of the afferent and efferent nerves, and of the center, for their efficient action.

The *cremaster reflex* is that drawing up of the testicle which is seen when the skin on the inner side of the thighs is irritated, and the *gluteal* when the skin of the buttocks is duly excited.

The *plantar reflex*, like the palmar, is produced by tickling the skin of the sole, and is seen in perfection when disease above cuts off the dorso-lumbar enlargement from the inhibition exerted by the brain.

The value of the reflexes has been carefully investigated by Knapp,* with the following results :

"Absence of the plantar or cremaster reflex is usually pathological, depending on a direct lesion of the reflex arc or some cerebral disturbance. Absence of the other cutaneous reflexes is not necessarily pathological."

The Deep Reflexes—The Knee Phenomenon—The Knee-Jerk.—The clonic movements produced by percussion of certain tendons have considerable pathological significance. If, when the knees are crossed, the patellar tendon is struck a smart blow with the ulnar side of the extended hand, or with the percussion hammer, a sudden extension of the foot takes place, caused by contraction of the thigh extensor muscles. This is called the "knee-jerk," and in health is very rarely absent. The explanation of this phenomenon is not yet agreed on, but by most observers it is regarded as a reflex. Although opinions may differ as to its nature, there can be no question of the value of this sign. In certain states of disease the knee-jerk is present in an exaggerated degree ; for example, when the cerebral inhibitory influence is withdrawn by disease of the pyramidal tract ; when an irritative state of the gray matter or of the efferent and afferent nerve fibers exists. The knee-jerk is absent whenever there are destructive lesions in any part of the reflex arc, as in posterior spinal sclerosis, in disease of the anterior cornua, and of the efferent fibers of the anterior roots, and when the inhibitory action of the cerebrum is increased.

The *ankle clonus* is another significant pathological sign, the presence of which can be made manifest in healthy persons only after preparation which has aptly been termed *sensitizing*. To induce this "sensitized" condition in healthy individuals, the leg is flexed on the thigh at an acute angle, the weight of the leg resting on the ball of the great toe, the heel raised from the floor. If now the top of the knee be struck a smart blow with the edge of the hand, and some voluntary motion be given to the limb in this position, a rhythmical movement, clonic spasm—the ankle clonus—will then go on independently. This movement is due to clonic contractions of the gastrocnemius, resulting in alternate elevation and depression of the knee and heel. In certain diseases, as lateral sclerosis, the ankle clonus is produced without preparation. If the heel is held in the operator's left hand, while the right puts the foot into the position of dorsal flexion by pressure on the ball of the great toe, the ankle clonus will then appear on tapping smartly the top of the knee. The clonus consists in rhythmical contractions and relaxation of the muscle, and consequent elevation and depression of the toes.

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* "Observations on the Cutaneous and Deep Reflexes." By Philip Coombs Knapp, A. M., M. D. (Harvard). "The American Journal of the Medical Sciences," April, 1885.

ACUTE AND CHRONIC NERVOUS DISEASES.

CEREBRAL HYPERÆMIA.

Definition.—*Cerebral hyperæmia*, or cerebral congestion, is a malady characterized by an increase in the amount of blood in the brain. The hyperæmia may be arterial, or *active;* venous, or *passive.*

Causes.—Any condition diminishing the amount of arterial blood in other parts will divert a larger quantity to the cranial cavity: compression of the abdominal aorta, ligation of an important artery, are examples. The suppression of an habitual discharge of blood—as that of hæmorrhoids, for illustration—is alleged to produce the same effect. Cerebral congestion occurs in the cold stage of an ague, and is also produced by the application of cold to the surface of the body. Prolonged intellectual effort, insolation, or sunstroke, protracted wakefulness, over-indulgence in alcoholic beverages, and the use of such narcotics as belladonna, are supposed to induce congestion of the brain. Hypertrophy of the heart, fullness of the general vascular system, and general plethora, are also alleged to have this effect, but grave doubts may well exist on this point. Passive congestion is produced when there is an obstacle to the return of blood from the cranial cavity, as when the superior *vena cava* and the jugular are compressed by intra-thoracic or cervical tumors, or when the venous system is overfilled by mitral or tricuspid disease. Venous stasis is also caused by atheromatous degeneration of the arterial tunics, feebleness of the cardiac contractions, and lowered vascular tonus.

Pathological Anatomy.—There are no structural changes beyond an increase in the amount of blood, the displacement of a corresponding amount of cerebro-spinal fluid, and mechanical compression of the cerebral matter. The veins of the dura mater are distended, but still more those of the pia mater and choroid plexus. The sinuses are also overfilled. The convolutions are somewhat flattened, and the perivascular lymph-spaces are closed by the approximation of their walls. On section, more blood than normal flows out of the divided vessels, and the *puncta vasculosa* are more numerous. If the hyperæmia is of long standing, or if repeated attacks have occurred, the changes are more pronounced. The veins enlarge and become varicose, and small arteries previously invisible come into permanent view, and aneurismal dilatations form on the arterioles. There may be minute extravasations and capillary hæmorrhages, the evidence of which is afforded in old cases by pigment deposits and blood-crystals in the lymph-spaces. Transudations of serum may occur in the subarachnoid spaces and in the ventricles, and also in the perivascular sheaths, whence it follows, in old cases, that permanent dilatation of these spaces may have occurred, producing the *état criblé.*

Symptoms.—There are three well-marked forms of cerebral hyperæmia—the *light*, the *severe*, and the *apoplectic* (Jaccoud). In the light form the onset is gradual, and among the first symptoms is headache, which is soon followed by characteristic signs : the headache is dull and heavy, with occasional sharp, lancinating pains, increased by motion or sudden shocks, or by light and sound ; there is inaptitude for any mental effort, and the attempt to exercise the mind causes a sense of cerebral exhaustion ; there is singing in the ears, with other subjective noises ; the conjunctivæ are injected, the retina is sensitive to light, and there are flashes of light and moving objects before the eyes ; the sleep is fitful and unrefreshing, and disturbed by dreams of a terrifying kind ; vertigo occurs, and the muscular movements are uncertain and fatiguing ; the sensations are disordered, and numbness and tingling are felt in the extremities ; the stomach is uncertain, and nausea is often experienced ; and the heart is exceedingly irritable, the pulse rising considerably with the least mental or physical effort or emotional excitement.* The *severe form* may develop out of the light, or it may come on without any prodromic symptoms. As compared with the light form, we find the headache is more intense ; the special senses are more irritable and intolerant of light and sound ; the mind more disturbed, ideation more confused, illusions and hallucinations occurring ; the wakefulness more obstinate and complete ; the motor functions more excited, the movements more irregular and uncertain, jactitations appearing ; the sensory functions are more perverted ; besides the headache, are neuralgic pains, especially in the fifth, numbness and tingling being felt in the extremities; the vertigo is more decided, the upright position being maintained with difficulty, and all coördinated and combined acts being executed with difficulty; the action of the heart is more excited, the pulsations irregular and rapid, and the least effort sending the beat up many times; the head is more decidedly warm, the eyes more suffused, more deeply injected, the eyelids more swollen; the stomach is more disordered, and nausea and vomiting are excited by effort of the mind, or by attempt at close attention. The symptoms indicate the approach of acute maniacal excitement, or acute inflammation; but the mind, although occupied by illusions and hallucinations, is still able to correct them or reason correctly about them, and the febrile condition does not yet exist. The symptoms may subside in a day or two, and health be restored in a few days, or, the case unrelieved may then pass into the stage of depression; torpor succeeding to exalted activity, drowsiness to wakefulness, coma to delirium. In adults, convulsions rarely occur in the course of the severe form, but are usual in children.

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* Hammond, "Cerebral Hyperæmia," p. 48.

In the apoplectiform variety of cerebral congestion, the patient may suddenly pass into unconsciousness, with the usual phenomena attending the apoplectic attack; there is complete muscular relaxation, involuntary evacuations may occur, but the reflex movements are not in abeyance, and in some minutes or hours the patient returns to consciousness, somewhat confused, however, and does not entirely recover for some days. Without losing consciousness, he may suffer confusion of mind, extreme vertigo, have defects of speech, or an entire loss of memory for words, numbness, tingling, and paresis of the members, nausea and vomiting, etc., also coming on suddenly, and disappearing after some hours and days without permanent disability. The symptoms belonging to the venous or passive form of hyperæmia are much less pronounced, although in some respects similar. There is headache, but a sensation of heaviness and dullness rather than acute pain; the eyelids are swollen and puffy, but the conjunctivæ are not injected; the superficial veins are full, but the scalp is cool; singing in the ears and impaired hearing are noted; vision is dull, and floating objects are seen before the eyes; the mental operations are dull and confused; somnolence passing into stupor, without continuous normal sleep, dreams, illusions, and sudden startings in the sleep, occur from time to time. On ophthalmoscopic examination, there are ascertained to be an enlargement of the retinal veins, more or less swelling of the optic disk, and vessels before invisible come into view. When the congestion of the brain is of the passive variety, the retinal veins are unduly enlarged and tortuous. Observations on the drum membrane disclose increased vascularity of this organ, which has intimate connection with the intra-cranial circulation. The superficial temperature of the head is elevated in active hyperæmia, but is not affected in the passive form. Surface thermometers and Lombard's thermo-electric pile are employed to ascertain the temperature of the scalp. In any case there will be but slight rise of the thermometer ; hence, any considerable elevation should awaken suspicion of inflammatory action.

Course, Duration, and Termination.—The light form may terminate in a few hours or days, under appropriate treatment, to recur from time to time, it may be ; or it may continue with fluctuations in the severity of the symptoms for months and years. A cure readily results, if the causes cease to operate and the right management is instituted. If the hyperæmia continue, other morbid conditions will arise out of it. The severe form has a variable duration. A cure may be effected if right treatment is instituted early enough, but structural alterations will not be long delayed, and mental derangement will occur at an early period, or a cerebral hæmorrhage may take place. The apoplectiform variety may terminate in health or in cerebral hæmorrhage, according to the method pursued and the nature of the causes. Attacks of this nature may precede cerebral hæmorrhage, as the author has several times witnessed, but they are not often repeated until the hæmorrhage takes place. The passive form pursues the fortunes of the lesions causing it, and hence the duration is very variable and the course protracted.

Diagnosis.—The symptoms being due to disturbances of the intracranial circulation, the diagnosis rests on the absence of symptoms indicating structural lesions—notably the absence of fever, the widespread bilateral diffusion of the symptoms, and the fugitive character of the attacks. It may be confounded with delirium tremens, epilepsy, apoplexy, stomachal vertigo, etc. As respects delirium tremens, the distinction rests on the habits, the previous history, and the severity and persistence of the symptoms in this disease. The attack of epilepsy is preceded by a cry ; then come pallor of the face, stertorous breathing from tetanic fixation of the muscles of respiration, cyanosis, and general convulsions. Children with congestion of the brain may have such convulsions as a symptom, but the history preceding and succeeding is very different in the two maladies. The apoplectic form is distinguished from apoplexy by the persistence of the reflex movements, by the absence of conjugate deviation of the eyes, and by the early recovery without hemiplegia. Stomachal vertigo is preceded by attacks of indigestion, and is accompanied by the conditions of syncope and anæmia, instead of hyperæmia.

Treatment.—Causes of the hyperæmia should cease, if possible. If it be the active form, the head should be elevated and cold applied, the feet being immersed in hot mustard-water. To withdraw temporarily from the circulation some of the blood, a ligature should be applied around the thigh or thighs for a time, alternating the application of the ligature to prevent injury. Leeches may also be applied to the mastoid process, or cups to the neck. In the apoplectiform variety venesection is advisable, as this is the most expeditious means of diminishing the intra-cranial blood-pressure. A brisk purgative is also an excellent expedient, relieving by acting as a derivative and by lessening vascular tension. The intra-cranial blood-pressure can also be lowered by the exhibition of veratrum viride, aconite, bromide of potassium, ergot, etc. These remedies are sufficient in the light form, but in the severe form a combination of the various means of treatment will be necessary. The treatment of the passive form is a part of the treatment required in the condition producing the hyperæmia, and need not now be discussed. The strictest attention must be paid to the diet and mode of life. An abstemious life—the diet consisting of fruit and vegetables chiefly—and early hours and the avoidance of all forms of excitement have prolonged life for many years, when an early demise was threatened by cerebral hyperæmia. Especially should alcoholic stimulants and the powerful emotions excited by speculations of all kinds be avoided. Such mild stimulants as tea and coffee even should be abandoned. In making these suggestions the author wishes his readers to note that he regards protracted rest to the mind as often injurious, and that light mental occupation is preferable to an entire disuse of the faculties.

CEREBRAL ANÆMIA.

Definition.—By *cerebral anæmia* is meant a lessened amount of blood in the brain. It may be *general* or *partial:* in the former the diminished supply of blood affects the whole organ; in the latter a particular district is deprived of its blood by the occlusion of a vessel. It is the general form of cerebral anæmia to be considered here.

Causes.—The most perfect type of cerebral anæmia is that produced by large loss of blood. Our knowledge of this condition has been rendered the more accurate by the experimental study of the subject in animals.* The effects of loss of blood on the functions of the brain are seen after severe hæmorrhage, as *post-partum* hæmorrhage, unavoidable hæmorrhage, menorrhagia, metrorrhagia, etc. Chronic wasting diseases, by the constant losses of nutrient material, induce cerebral anæmia. Phthisis, chronic dysentery, suppuration, and prolonged lactation, belong to this category. Maladies which impair the power to produce nutrient material, affecting the primary and secondary assimilation, will also cause anæmia of the brain. To this state as it occurs in infants was applied the term *hydrocephaloid* by Marshall Hall, who first demonstrated the important fact that a condition supposed to be due to inflammation was really the product of anæmia. Under the influence of shock, by powerful mental or moral emotions, a sudden

* Kussmaul and Tenner, "Sydenham Society's Translation."

contraction of the intra-cranial vessels occurs, and syncope, with loss of consciousness, ensues. Feebleness of the heart induces anæmia of the brain—a fact well exemplified in the sudden pallor and faintness experienced by convalescents on rising up after long decubitus; also in the case of those who suffer from weak heart, fatty heart, or obstruction at the aortic orifice, etc.

Pathological Anatomy.—The morbid changes are very simple. The amount of blood is below the normal, and the vessels are less full. The appearance of the brain is pale and exsanguine, and on transverse section of the hemispheres there are no bloody points. The subarachnoid spaces and the ventricles contain a good deal of fluid, and the perivascular lymph-spaces are also well filled with fluid, for, as the vessels contain less blood, the cerebro-spinal fluid increases; while in hyperæmia the distention of the vessels forces the fluid out, closes the lymph-spaces, and flattens the convolutions. The opposite state obtains in anæmia: the brain is pale, white, and moist; the vessels small, the lymph-spaces large. In partial anæmia, other factors are concerned, and hence the local conditions differ.

Symptoms.—There are two distinct forms: *acute*, or sudden; *chronic*, or light. Venesection *ad deliquum animi* furnishes a complete picture of the first: the face grows deadly pale, the lips white, the pupils dilate, the action of the heart becomes very feeble, the pulse small, a cold sweat breaks out over the body, ringing noises sound in the ears, surrounding objects appear dim, and a mist gathers before the eyes; voices are heard in the distance, and the words are unintelligible, everything fades suddenly out of consciousness, and the patient falls as if lifeless, respiration having ceased, and the heat-beart scarcely continuing. There is complete muscular resolution, but in an instant the eyelids begin to tremble, the muscles of the lips and face twitch, and a general convulsion follows. The syncope, which is merely a fainting-fit, does not proceed any further than suspension of consciousness, and in a short time the respiration begins, the heart-beat grows stronger, the patient opens his eyes, looks around with a dazed expression, and asks what has happened; he tries to get up, and finds himself very weak, but in a short time the bodily vigor is entirely restored. The convulsions of cerebral anæmia are due to two factors: to an abnormal excitability of the "spasm-center"; to the circulation of black blood through this spasm-center. In the slow, habitual, or chronic anæmia, the condition is that of depression of function. The brain, inadequately supplied with nutrient material, functionates imperfectly; the special senses are both irritable and depressed—the sight is dull (amblyopia), and light is painful to the eyes; hearing is obtuse, there are subjective noises in the ears, *tinnitus*, etc., and loud sounds are distressing; the mental operations are slow and confused, and there may be illusions, hallucinations, maniacal excitement, etc. (puerperal mania, insanity of lactation, etc.); muscular movements are excited, or depressed and feeble, tremulous or incoördinate; the sensory functions are similarly affected—there may be excitement or depression, neuralgic pains, numbness, prickling, tingling, or anæsthesia; vertigo is nearly always present, and consequent uncertainty of movements; headache is also commonly present, and may be a sense of heaviness or oppression, or, more frequently, acute pain; exertion causes great fatigue, and syncopal attacks are easily induced; the action of the heart is weak, and rapid action is excited by the least movement; and the sense of faintness is usually accompanied by nausea. In the form of cerebral anæmia, known as hydrocephaloid, the child is exhausted by a wasting malady; its surface is cool, skin pale, the pulse quick and weak, the eyes are half closed, sunken, and surrounded by broad, dark areolæ, the fontanelle is concave, the head cool; there is much fretfulness, although there is a somnolent state; the stomach is irritable, the bowels relaxed.

Course, Duration, and Termination.—The acute form, so far as the immediate attack is concerned, lasts a few minutes only, but this is merely a symptom of a long-established anæmia of the brain. The chronic form has an indefinite duration, and pursues a varying course according to the management and the nature of the causes. The termination is usually in restoration to the normal state, if the treatment be suitable. So important are the changes in the vessel-walls in anæmia, that we should not overlook the gravity of any case that has continued a long time. Furthermore, as various intercurrent maladies may develop, prognostic opinions should be expressed with caution if the anæmia has persisted.

Diagnosis.—As cerebral hyperæmia presents many symptoms in common with cerebral anæmia, the diagnosis of these affections may be confused, but attention to a few points ought to conduct to right conclusions. The history of the causes, the appearances of anæmia, and the depression of the circulation, will indicate the nature of the case. The use of the surface thermometer, or thermo-electric pile, to ascertain the temperature of the scalp, is necessary, for in anæmia the temperature is rather below than above normal, but in hyperæmia the opposite condition obtains. Ophthalmoscopic inspection of the retina and otoscopic inspection of the drum membrane should be made, to ascertain the character of the circulation: in hyperæmia the retinal vessels are abnormally full and the drum is red and injected, whereas in anæmia the retina and drum membrane are pale and comparatively bloodless.

Treatment.—The recumbent posture and stimulation of the nares with ammonia are the only measures necessary in the treatment of syncope. When alarming depression is due to hæmorrhage, besides the measures necessary to stop the loss of blood, anæmia of the brain is to be overcome by depression of the head and elevation of the limbs, by the administration of alcoholic stimulants, by the subcutaneous injection of stimulants, by the intravenous injection of ammonia, and by transfusion. The chronic form of cerebral anæmia is to be arrested by stopping the sources of waste, by the use of iron and the phosphates, and by judicious alimentation. The best results are obtained by the administration of a stimulant to the cerobro-spinal axis (strychnine) and a chalybeate tonic. Arsenic is often highly serviceable in cerebral anæmia, in combination with iron. For the maniacal delirium of cerebral anæmia, the hypodermatic injection of morphine is of the greatest value. When there is associated with this delirium a high degree of motor excitement, atropine or duboisine should be combined with the morphine.

OCCLUSION OF THE CEREBRAL VESSELS.

Definition.—Under this term are included all lesions which occlude or block the vessels, thus causing anæmia of some part or parts of the brain. The occlusion may form in a cerebral vessel, or may be produced by an embolism conveyed thither from any part of the vascular system. Under this term must be comprised the remote as well as the immediate results of occlusion.

Causes.—The factors chiefly concerned in the occlusion of intracranial vessels are thrombosis and embolism. Chronic endarteritis and slowing with weakening of the blood-current are the causes of thrombosis. The changes in the arterial tunics consist in atheromatous and calcareous degeneration ; the lumen of the vessel is gradually narrowed by the deposition of new material, and the intima is roughened. The propulsion of the blood is hindered by weakness of the heart's action, and by diminished elasticity of the walls of the arteries, due to the atheromatous changes in the tunics. When the disease in the walls of a cerebral vessel reaches a certain point, coagulation of the blood takes place and an occlusion (*autochthonous thrombosis*) is thus effected. The formation of a thrombus is also favored by the condition of the blood itself. In chronic wasting diseases, the relative proportion of fibrin in the blood being much increased, coagulation is promoted accordingly. An autochthonous thrombus may form in a vessel whose lumen had been obstructed by the pressure of a tumor.

Emboli consist of bits of fibrin, exudations, or concretions, which, formed at some distant point and carried into the circulation, are deposited in the brain. The most usual source of emboli is endocarditis, either of the ulcerative variety or of the chronic form with its polyp-like excrescences, or fibrin vegetations. According to the observations of Bertin, the emboli come from the left auricle, four times ; from the left ventricle, twelve times ; from the aortic valves, ten times; from the mitral, twenty-four times. These figures agree with the usual experience on this point. Cardiac emboli are also produced in the following way: clots form, especially in the auricle, when the heart is weakened by myocarditis, fatty degeneration, uncompensated valvular lesions, and such chronic wasting diseases as cancer and tuberculosis. Such clots, subsequently pulverized by the cardiac movements, are carried into the circulation. Emboli may also be derived from aortic aneurism, from syphiloma of the great vessels, etc.

Pathological Anatomy.—Owing to its position at or near the summit of the arch of the aorta, the blood-current from the aortic orifice is directed to the left common carotid, so that an embolus loosened from the heart naturally enters this vessel, and its prolongation within the cranium, the Sylvian artery. It necessarily follows from this that the left side is usually obstructed. It rarely happens that an embolus enters the vertebral arteries. Sometimes the embolisms are multiple, and enter the vessels on both sides, or are lodged in different places on the left side. As certain vessels are usually occluded, it is important to have a clear understanding of the parts supplied by them. The left Sylvian artery sends branches to the second and third frontal convolutions, the anterior and superior portions of the three temporal convolutions, the island of Reil, the parietal convolutions, part of the external and all of the internal capsule, the lenticular nucleus, and most of the corpus striatum. It is important to note, further, that the vessels of this part of the brain have the arrangement of Cohnheim's terminal arteries—arteries without anastomoses—while the vessels of the gray matter of the hemispheres, or the cortex, communicate freely with each other.* When an artery of the "basal system" is obstructed, either by a thrombus or embolism, an anæmia of the territory supplied by the vessel at once ensues—either a simple anæmia and white softening, or anæmia followed by collateral hyperæmia and œdema. The simple anæmia and white or yellowish-white softening occur when the blood in the whole extent of the occluded vessel coagulating, prevents the backward flow of blood through the capillaries, and consequently the collateral hyperæmia and œdema. The anæmic tissue dies or undergoes *necrobiosis* in consequence of the loss of its entire nutritive supply. The nerve-tissue elements become disassociated, break up into a diffluent granular mass, and are crowded with fat-cells, whence the color of the softened tissues assumes a somewhat yellowish aspect. Yellow softening is also a stage of the next form. When a terminal artery is occluded, and all parts of the vessel beyond the seat of obstruction remain pervious, blood flows back through the capillaries from the nearest artery and vein, until the previously anæmic and bloodless district is deeply engorged. Changes now occur in the walls of the vessels, permitting diapedesis of the red blood-globules. As, in the process of softening and disintegration which now ensues, the tissues are colored by the red corpuscles, the appearances are entitled "red softening." Minute extravasations occur here and there, from rupture of capillaries, and hence, in the midst of a uniform red there will be seen the dark points of "capillary apoplexy." These extravasations may be so numerous as to present the appearance of a cerebral hæmorrhage. In from two to four weeks the red softening becomes yellow softening in consequence of the transformation of the hæmoglobulin and the fatty degeneration of the nerve-elements. The softening proceeding to another stage becomes "white softening," when there is a milky, or rather creamy fluid, containing, mixed with it, masses or particles of broken-down nerve-elements. There is no abrupt line of demarkation, but the diseased part shades off into the surrounding healthy part by a fine gradation.

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* The reader should peruse in this connection the articles on "Arteritis" and on "Thrombosis and Embolism."

Symptoms.—There are two well-defined modes of onset: the gradual, which occurs to thrombosis; the sudden, or apoplectic, due to embolism. The first form, or thrombosis, is a malady of the old; the second form, or embolism, may occur at any period, frequently in the young. As, when chronic arteritis of the cerebral vessels exists, a number of them may be diseased at the same time, the resulting symptoms must necessarily be widely diffused, and, as the disease has proceeded to different stages at different points, there may be present, at the same time, the symptoms of excitation and depression of function. Headache, more or less persistent, and of variable intensity, is the earliest symptom; next, alterations of character become evident—the individual grows irritable, morose, and despondent, his mind is easily fatigued, and memory is impaired; at first names, then some unusual word, ultimately most words, are forgotten. Occasionally the only mental defect observed is loss of the memory for words—amnesia of verbal language—which may occur slowly or suddenly, with or without something of a stroke. After the headache, vertigo comes on, and may be occasional and caused by a change of posture, or it may be constant when sitting up and when recumbent. Difficulty of locomotion is experienced, in consequence partly of the vertigo, but chiefly because of weakness of a group of muscles or of a member; more or less of senile trembling may be present, or the trembling of muscular weakness; and the movements of the tongue may be imperfect and speech hesitating and mumbling. There are two causes for the symptoms just detailed—gradual encroachment on the lumen of diseased vessels, whence the blood-stream is lessened, and interference with the nutrition of the brain by reason of calcareous degeneration of the capillaries. The next point in the morbid complexus is the occurrence of a sudden attack, which may or may not be apoplectic. If apoplectic, the patient falls suddenly into a condition of insensibility, with complete muscular resolution. On emerging from such an attack there may be hemiplegia; if right hemiplegia, associated with more or less disability of speech, possibly with aphasia. In other cases, with equal suddenness, but without any apoplectic seizure, there may occur a hemiplegia, or the paralysis may be limited to the arm, or to the leg, or to the face; it may be complete or partial (paresis), and with weakness there may be contractions and rigidity. The paralysis may disappear quickly, and after an uncertain period may occur again, or be succeeded by rigidity and contraction. The disappearance of a paralysis under these circumstances means the reopening of the obstructed area to the circulation by collateral channels or anastomoses—a condition of things only possible in the cortex. An autochthonous thrombus may form in a vessel of the basal system. The final occlusion of the vessel may be preceded by various prodromata—by headaches, vertiginous sensations, numbness, tingling, formication, coldness, muscular cramps, etc. Paralysis may develop slowly, as the thrombus slowly forms, or suddenly, with the usual phenomena of the apoplectic stroke; the paralysis is strictly localized and does not change, for, the vessels being of the terminal kind, collateral hyperæmia and œdema result, and the affected tissue goes through the process of necrobiosis. When occlusion occurs in this way, the subsequent phenomena are the same as those of embolism. As the embolus causing the cerebral mischief comes from some distant point in the vascular system, it is obvious that there can be no intra-cranial disorders produced by it ere it effects a lodgment in the brain. It is evident that there must be very considerable variation in the severity of the symptoms, according to the importance and the situation of the vessel occluded. In a majority of cases the attack is apoplectic—there may be for an instant intense headache and dizziness, sudden flush or pallor of the face, or the patient may utter a wild cry—he falls immediately into unconsciousness, with complete muscular resolution, or there may be a distinct epileptiform seizure. Instead of unconsciousness, the stroke may be nothing more than a severe vertigo, with confusion of mind, muscular twitchings on the affected side, and vomiting. Vomiting may also occur in the apoplectic form, just as the mental confusion is coming on. On recovering from the stroke or shock—which is doubtless due to the suddenly produced partial anæmia, effecting at the same moment an immense change in the intra-cranial blood-pressure—a hemiplegia is found to exist, and it is most frequently of the right side, owing to the arrangement of the vessels on the left side of the brain. Although right hemiplegia is usual, it is not invariable: there may be left hemiplegia, or bilateral paralysis, or paralysis of the different cranial nerves. Embolism may also affect the central artery of the retina, and amaurosis result from the occlusion. Double optic neuritis arises during the course of all "coarse organic lesions" of the brain, and hence ophthalmoscopic examination is a necessary duty in such cases. The mental functions are variously affected. In the slow form of occlusion—thrombosis from chronic endarteritis—there is gradual mental failure, beginning in loss of memory, and thence the spectacle of senile dementia. In embolism the mental faculties are, during the period of coma, entirely suspended; if the patient emerge from this with hemiplegia, the mind is always enfeebled to a greater or less extent, the language faculty is variously impaired, the emotional nature is highly excited, and the reason and judgment are clouded. With right hemiplegia from embolism there is usually associated *aphasia*, or loss or impairment of the faculty of communicating ideas by words or by signs. The hemiplegia involves the tongue and the corresponding side of the face. The reflex movements are readily excited in the paralyzed parts. When there is embolic obstruction of the basilar artery, the symptoms differ somewhat from the description above given. The hemispheres are not involved, nor the important parts supplied by the Sylvian artery; there is no apoplectic seizure, nor loss of consciousness, nor troubles of the intellectual faculties. There are disorders in vocal expression, due to paralysis or ataxia of the muscles of the tongue (ataxic aphasia), but vertigo and vomiting are usual symptoms.

Course, Duration, and Termination.—The course of symptoms referable to the changes preceding and resulting in thrombosis is essentially chronic. Months and years may be occupied in reaching the point of coagulation, and other months, even years, may be passed in the paralytic state. When the lesions are of the basal system they are permanent. Although there may be some improvement, which, however, does not continue, the members paralyzed remain in the condition at which they had arrived after several months. In thromboses the most sudden and considerable improvement takes place in paralysis of members, defects of speech, and disorders of sensations, due to disease of the vessels of the cortex; but the probability of the return of these lesions, or of the appearance of other lesions, should not be forgotten. While the prospect of great immediate improvement is good in such cases, the future must be regarded with apprehension. On the other hand, in embolic occlusion, the immediate results are more severe. Death may be the result of the occlusion of a large vessel within two or three days, or longer, the patient never emerging from the coma. In other cases the patient arouses from the coma, hemiplegia exists with aphasia, the temperature rises a little as the collateral hyperæmia and œdema come on, but falls again in a few days, and the case then pursues the usual course of localized softening from any cause. Right hemiplegia and aphasia, from blocking of the left middle cerebral, may occur in youth, early manhood, at any period in fact, and are associated with valvular disease, usually of rheumatic origin. These lesions may also be associated with aneurism, with syphiloma, or with ulcerative endocarditis.

Diagnosis.—The diagnosis of thrombosis rests on the evidence of chronic arteritis—the simultaneous presence of the changes in the radial, the color of the hair, the condition of the skin, an arcus senilis; on the variability and diffusion of the prodromal signs, and those of the established lesions. Embolism is known by the age of the subject (often so at least), by the history of rheumatism, the existence of valvular lesions, by the suddenness of onset without prodromes.

Treatment.—The author has had remarkable results from the following plan of treatment in thrombosis: Carbonate and iodide of ammonium (ten grains of the former and five grains of the latter) are given three times a day in a suitable vehicle, for several months, usually, the object being dual—to increase the action of the heart and arteries, and to effect a solution of thrombi forming by maintaining the alkalinity of the blood. To postpone and possibly arrest the atheromatous degeneration of the vessels, cod-liver oil and the sirup of the lactophosphate of lime are regularly exhibited (a teaspoonful of each) three times a day, immediately after meals. The ammonia solution is administered before meals. At the same time these remedies are being given, a daily dose (at 10 A. M.) of quinine (five to ten grains) is also prescribed, should there be a condition of depression and languor of the intracranial circulation requiring it, but the carbonate of ammonia is usually sufficient. With this plan is conjoined a suitable regimen—a simple but nutritious diet, moderate exercise, and careful supervision of the various excreta. As soon as possible after an embolic obstruction has occurred, carbonate of ammonia should be given—very usefully in the liquor ammonii acetatis—and should be kept up for weeks. The most absolute rest should be maintained, and the diet should be light and unstimulating. In a month or two a very light galvanic current (from two cups) may be passed through the brain in both directions. Quinine is most useful, especially if there be any elevation of temperature; but in all cases it has seemed to the author highly useful after some weeks' administration of ammonium carbonate.

OBLITERATION OF THE CEREBRAL CAPILLARIES.

Pathogeny.—The capillaries of the brain are occluded by the finer particles which readily pass through the larger vessels. In the severer forms of acute malarial poisoning small particles of pigment are formed, and, entering the cerebral capillaries, lodge, and are known as "*pigment embolisms*." Violent delirium, terminating in coma, and sometimes convulsions, may result from the occlusions formed in this way. The white-blood corpuscles, under conditions not now understood, aggregate in masses and form emboli. These are probably examples of pyæmic change, for such emboli have been formed in connection with pyæmia, erysipelas of the face, etc. Emboli, consisting of particles of cancerous, septic, or decomposing material—*infective emboli*—may also be minute enough to pass the larger vessels and occlude the cerebral capillaries. In very rare cases the capillaries are blocked by lime salts, taken up at some point where disintegration of bone is going on—*lime-salts emboli*. Again, emboli consist of fat-globules which enter the blood from the marrow of fractured bones—*fat emboli*. The capillaries of the lungs may arrest them entirely, and hence the most serious symptoms are referable to these organs; but the finest globules may pass through the lungs and block some of the cerebral capillaries. As the anastomoses between the capillaries are very abundant, it is obvious that if the obstructions are but few in number they will be compensated for. When numerous, there will be produced anæmia, followed by the usual changes of necrobiosis, ending in softening.

Symptoms.—In the case of pigment embolisms occurring during a malarial fever, the onset of this malady is announced by intense headache, vertigo, delirium, sometimes convulsions, and the febrile phenomena are greatly intensified. If, during the course of facial erysipelas, similar symptoms arise, they may be due to white-corpuscle embolisms, or, if occurring after a fracture of a bone, may be due to fat-embolisms. When the embolisms are not very numerous the symptoms may be less pronounced: there may be dizziness, loss of memory, and other mental defects, persistent headache, etc. In any case the diagnosis can hardly be more than a fortunate guess. The treatment may be conducted on the same basis as that of occlusion of the arteries.

OCCLUSION OF THE CEREBRAL SINUSES.

Pathogeny.—Thrombosis is the mode of occlusion of the cerebral sinuses, and it may result from venous stasis or from phlebitis. In the former case the propelling power of the heart is much reduced, and the fibrin of the blood increased (hyperinosis). This condition of affairs occurs chiefly in children exhausted by long-standing illness; in the cases observed by the author, there had existed an ileo-colitis of several weeks. The phlebitis is secondary to some morbid process in the neighborhood, most frequently to caries of the petrous portion of the temporal bone, and the petrosal or transverse sinus only may be attacked, but the purulent phlebitis extends occasionally to the cavernous sinus and the circular sinus. Next to caries of the bones, the most frequent cause of this form of thrombus is erysipelas of the head and face, carbuncle of the upper lip or nose, and malignant pustule of the lip. The position of the thrombus is determined by the nature of the cause: if caries, the thrombus is found in the transverse or petrosal or cavernous sinus; if erysipelas, or malignant carbuncle, in the pterygoid plexus and cavernous sinus ; if stasis from cardiac feebleness and hyperinosis, in the longitudinal sinus. The thrombus and the subsequent changes taking place in it are the same as those already described. The vessels entering the sinus, the seat of occlusion, are turgid, tortuous, and their tunics weakened, so that they yield to the increased pressure, and hæmorrhages occur at various points, on the hemispheres, especially in the cortex. Softening occurs to a small extent about the hæmorrhagic extravasations, and meningitis may arise as a complication.

Symptoms.—As the cases of thrombosis of the sinuses occur in the subjects of wasting maladies, or of cardiac feebleness, the symptoms produced by the thrombus are superadded to those of the original malady. The signs by which such an occurrence may be recognized are all the more obscure, since the anæmia of the brain may be accompanied by many of them. There have been observed the following: rigidity of the cervical muscles, the occiput being buried in the pillow, and sometimes general muscular rigidity ; ptosis, strabismus, nystagmus, and paresis of facial muscles ; hebetude of mind, stupor passing into coma, sometimes delirium ; headache, vertigo, nausea and vomiting ; delirium, ending in coma ; contractures, or paresis, local tremor, clonic convulsions ; paralysis may be crossed with contractures and rigidity. Indeed, so various and diffused are the symptoms that the diagnosis must always be in the nature of a guess. More importance is to be attached to circulatory disturbances affecting external vessels. The facial vein communicates with the pterygoid plexus of veins and the cavernous sinus ; the nasal veins communicate through the foramen cæcum with the longitudinal sinus, and the occipital veins communicate with the transverse sinus by the *emissaria mastoidea*.* Hence, bleeding at the nose, puffiness of the eyelids, swelling of the facial vein, and of the occipital veins, accompany thrombosis of the sinuses. From the same cause there will be prominence of the eyeballs, injection of the conjunctivæ, and a swollen and tortuous condition of the retinal veins, cloudy swelling of the optic disk (choked disks), etc. In the case of thrombus of the cavernous sinus, there may be irritation by pressure of the fifth nerve, and consequent neuralgia—of the fourth, and internal strabismus ; of the oculo-motor, and contracted pupil and external strabismus, etc. These symptoms have a high degree of importance if present ; but their absence does not negative the existence of thrombosis. During the course of chronic otorrhœa and caries of the petrous bone, cerebral symptoms may supervene, and a fever of septicæmic character develop. When delirium tending to coma accompanied with typhoid symptoms appears during erysipelas or phlegmon of the upper lip, there may be suspected, as in the former case, that the new symptoms may be due to thrombosis of a sinus. The diagnosis must always be largely conjectural.

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* Henle, "Gefässlehre," p. 341.

Treatment.—The treatment consists in the free use of carbonate of ammonia and quinine, given with the objects in view indicated under the head of occlusion of the cerebral vessels. Unfortunately, when this accident occurs, there is little chance of accomplishing any good. Whenever a phlegmon of the upper lip appears, the probability of this accident should be kept in view. Free administration of quinine is undoubtedly serviceable in preventing this complication.

CEREBRAL HÆMORRHAGE.

Definition.—By this term is meant, the giving way of a vessel and the escape of blood into the cerebral tissues. *Apoplexy* is sometimes used synonymously with cerebral hemorrhage, but incorrectly, since it is a symptom merely, and not a disease.

Causes.—The principal cause of cerebral hemorrhage is disease of the vessels—aneurismal dilatations seated on the arterioles and varying in size from a pin's-head to bodies too minute for the unaided sight to recognize. It is rare for these bodies to form before forty, but they occur with increasing frequency with the advance in life. The change is a periarteritis and begins in the perivascular lymph-sheaths, thence extends to the adventitia, the muscular layer dilates, and the aneurism is formed.* Atheromatous degeneration of the tunics of the vessels may be an indirect cause, by leading to the formation of the miliary aneurism. Increase in the blood-pressure is said to have an influence in causing hæmorrhage, but not directly. When disease has weakened the vessels, an increase in the blood-pressure will cause them to yield, but, without such change in the walls of the vessels, mere variations of pressure will not suffice. The principal source of increased blood-pressure is hypertrophy of the left ventricle—that form associated with hypertrophy of the muscular layer of the arterioles and contracted or fibroid kidney. Besides the constantly exalted pressure, the intra-cranial vessels may be exposed to sudden increased strain by a variety of causes : by stimulants, as alcohol, opium, coffee, tea, etc. ; by a cold or hot bath, by a full meal, and by moral emotion. Cerebral hæmorrhage is notably increased by the cold weather of autumn. Venous hyperæmia may lead to cerebral hæmorrhage, as coughing, straining at stool, coitus, etc., but disease of the vessel-walls must predispose to the accident. The arterial disease on which hæmorrhage depends is probably transmissible, for it is a matter of common observation that the tendency to cerebral hæmorrhage is inherited.

Pathological Anatomy. — Certain parts of the brain seem particularly liable to cerebral hæmorrhage : the corpus striatum, the lenticular nucleus, the thalamus opticus. When these parts are affected, the damage is not always confined to them, but the neighboring parts of the hemisphere are damaged simultaneously, and the lobes of the hemispheres are often separately attacked, the anterior and middle more frequently than the posterior lobe. Next in point of frequency, but much less often, the cerebellum is involved, and lastly, although rarely, the pons and medulla. The blood is not necessarily confined to the point whence it escaped : it may break through to the surface or into the ventricles and pass by the *iter* from the third to the fourth ventricle. When the amount is large, the dura mater may be put on the stretch, the convolutions compressed, the sulci lessened in depth. The blood may be collected in a mass or focus, or it may be spread out into a more or less thin layer. When in a focus, as is most usual, the collection is somewhat circular and varies in size from a pea to an English walnut, or larger. There may be one or several foci, and they may occur in symmetrical parts—as a focus in each corpus striatum, for example. Besides a recent there may remain the evidences of former hæmorrhages. Immediately after it has occurred there is a blood-clot, dark in color and homogeneous in its constituents, which are those of blood merely, although around it is broken-down cerebral matter, mixed with blood-clot, and in the mass somewhere will be found, if carefully traced out in water, the affected vessel and its ruptured miliary aneurism. Soon after the clot has formed, separation begins, and the fibrin collects in the center of the mass or at the periphery, while the corpuscles adhere in a group, and the serum pressed out saturates the adjacent broken-up cerebral matter. The next step, if death does not occur, is the retrograde change in the blood-clot, which becomes first of a dark chocolate-color, but the hæmatin disappears, the watery part is absorbed, and a yellow, puriform-looking material only remains. A limiting inflammation may occur in the adjacent cerebral matter, a connective-tissue membrane of a spongy structure forms, and the remains of the clot will be inclosed in this. Besides the yellowish, puriform fluid or a whitish, whey-like fluid, there are contained crystals of pigment in the meshes of the cyst-walls. The clot and the surrounding brain-substance do not always undergo this favorable disposition. An inflammation may be lighted up in the brain-tissue, around the clot, in a few days after it has formed, producing extensive softening and œdema. The cysts formed may continue indefinitely without further change, or they may ultimately disappear, leaving only a cicatrix of considerable area, but thin, and composed of either dense connective tissue, or of a spongy material containing pigment. The changes due to cerebral hæmorrhage are not limited to the site of the original injury. Some months afterward an atrophic degeneration has taken place in the nerve-fibers of the pyramidal tracts. These degenerative changes do not follow all cases of cerebral hæmorrhage. They occur after hæmorrhage into the internal capsule, the corpus striatum, the gray matter of the motor zone, and the subjacent white substance, and less so when the lesion is in the optic thalamus and centrum ovale, and not at all when the hæmorrhage is in the caudate nucleus.* The atrophy extends downward through the *crus*, the *pons*, and the pyramidal tracts, and consists in wasting of the nerve-elements and an increase of the connective tissue.

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* Eichler, "Deutsch. Archiv für klinische Med.," xxi, 1, 32.

Symptoms.—Many cases of cerebral hæmorrhage are preceded by distinct prodromes. The most usual are those connected with chronic arteritis, which may lead to thrombosis, or less frequently those dependent on cerebral hyperæmia. Headache, vertigo, sudden attacks in which the mind is confused, the memory for words is lost, or mistakes in the use of words occur; changes in the disposition, becoming morose, dejected, and irritable, weakness of a limb or of one side, numbness, tingling, or a feeling of coldness in a member or several members, double vision, weakness of the tongue, paresis of the facial muscles, etc. Sometimes, as the author has witnessed, the apoplectiform variety of cerebral congestion is followed in a few weeks by severe or fatal cerebral hæmorrhage. In many cases there are no "warnings," no prodromata, but the hæmorrhage occurs suddenly. The character of the seizure varies greatly. It may be apoplectic; the patient utters a cry or a groan, and falls insensible. Usually some symptoms occur just previously to the loss of consciousness; there is headache of a very intense kind, or giddiness with nausea and vomiting, or the tongue is paralyzed and speech impossible, or there is delirium or incoherent rambling, or there is gaping, a feeling of great desire for sleep, and increasing drowsiness, or there may be intense weakness of the limbs and a feeling of exhaustion, or one limb may be seized with intense numbness and tingling, or there may be spasm of the muscles soon to be paralyzed—in a great variety of ways the attack may be announced some hours or minutes before the blow falls. The patient passes into unconsciousness, with complete muscular relaxation, and the extinction of reflex movements, the action of the heart and the respiration continuing. In the less severe cases the unconsciousness is profound, but strong irritation may induce reflex movements, and swallowing is possible if the substance is placed in the pharynx, and a difference between the movements of the two sides is also apparent. The eyes—and the head, also, frequently—deviate toward the side affected in the brain and from the side paralyzed: this movement constitutes a means of diagnosis between cerebral hæmorrhage and other causes of profound unconsciousness. Convulsions of the epileptiform variety may occur, when the hæmorrhage causes unconsciousness, and usually signifies large hæmorrhage, or hæmorrhage into the pons or medulla. When the hæmorrhage occurs slowly, and the patient glides gradually into unconsciousness, there may be nausea, vomiting, and pallor of the face, but in most cases of cerebral hæmorrhage the face is rather red and flushed. There is no constant rule as to the size of the pupils: a very minutely contracted pupil usually signifies hæmorrhage into the pons; and unequal pupils, one being largely dilated, indicate a large hæmorrhage breaking through into the lateral ventricle. The breathing has usually, but by no means invariably, the stertorous character, by which is meant the drawing in of the paralyzed cheek with inspiration and its puffing out with a sort of explosion in expiration. The pulse is small or full, slow or irregular, usually slow and full. There are apoplectic examples of cerebral hæmorrhage in which the unconsciousness is not profound—the patient may be roused, if he is loudly called, but lapses into a soporose state at once. There are many cases in which consciousness is not lost at all: there may be a temporary confusion, or some of the symptoms called prodromal, and then paralysis of one side occurs. Often it is sudden and complete; again it comes on slowly, and is not complete for some minutes. In the apoplectic form, death may occur during the unconsciousness—in from five minutes to three days. The fulminant cases, which terminate in a few minutes, are comparatively rare—sudden death being usually caused by heart-disease. If unconsciousness continues longer than twenty-four hours, death is the usual result. The temperature during the period of unconsciousness is low—below the normal, one or two degrees—but at the end of the first day a rise to normal or a little above takes place, and, if a fatal result, there is a great rise just before death. Pneumonia is apt to be the cause of death, especially when the cerebral lesion is somewhere in the right hemisphere, as Brown-Séquard has demonstrated. Consciousness may return in a few minutes, but usually in from half an hour to three hours. Again, the effects of the seizure may continue for days, there being stupor, confusion of mind, defects of speech. The return of consciousness is indicated by the revival of reflex excitability, by the effects of irritation, etc. The progress of restoration may be retarded by the onset of inflammatory symptoms at the expiration of two or three days; the temperature rises a degree or two; headache, confusion of mind, and delirium occur; tonic contractions ("early rigidity") ensue in the paralyzed muscles, and they become the seat of severe pain, which may persist for a month or more, while the other symptoms disappear in a few days.

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* Flechsig, "Archiv für Heilkunde," 1877, and No. 53, 1878.

When the disturbances due to the seizure subside, then may be clearly seen the extent of the paralysis. The shock of the attack suspends the functions of many parts of the cerebrum, which soon functionate again as these effects of the injury subside. Various paretic and paralytic symptoms, that appear at first, quickly cease, but the more permanent results are the more evident. The amount of paralysis varies from a hardly appreciable weakness to an absolute extinction of motility. As there is usually but one focus of hæmorrhage, the resulting paralysis is *unilateral*, and on the side opposite the lesion, and involves the muscles of the face, of the tongue, of the body, and of the extremities—*right or left hemiplegia*—according to the cerebral hemisphere invaded. The muscles of the face paralyzed are those of expression, and are innervated by the seventh nerve. Those branches of the nerve distributed to the orbicularis palpebrarum, corrugator supercilii, and the frontalis are but slightly affected, the labio-nasal fold is flattened or obliterated, and the corner of the mouth is depressed. The tongue when protruded deviates toward the paralyzed side, and the palate may hang lower than normal and turned toward either side. In consequence of the paralysis of the expression muscles, many movements become awkward or impossible, as whistling, pursing up the mouth, laughing, etc. The muscles of the chest are paretic, and respiration somewhat hindered thereby (Nothnagel). The extensors seem to be more affected than the flexors, but this is only apparent, because of the greater power of the latter. Notwithstanding the immense preponderance of cases proving the crossing of the motor fibers, and consequently the occurrence of hemiplegia on the side opposite the seat of the lesions of the brain, there are opposing observations. Bilateral paralysis may be due to simultaneous lesions on both sides, and in this way bilateral hemiplegia may be produced. Paralyses are said to be "alternating" or "crossed" when the paralysis of the face is on one side and of the extremities on the other. This may occur in lesions of the pons, etc. Although the paralyzed parts may be motionless, they may execute "associated movements": thus, in coughing or sneezing the paralyzed member may give a jerk, or may imitate movements performed by the healthy side. The contractions which accompany the hæmorrhage, or which are excited by an inflammatory process about the site of the clot in a few days after the seizure (early rigidity), have already been referred to. The contraction which occurs later, after the paralysis has existed for a long time, is known as "late rigidity," but its intensity and persistence bear no constant relation to the character of the case, except its duration, and rigidity may not be present at all, although not often absent. Bouchard's explanation that the rigidity depends on the atrophic descending changes in the pyramidal tract continues to be the most generally accepted theory of this phenomenon, but it is not altogether satisfactory. Besides rigidity, long-paralyzed members may be affected by choreic movements, first described by Wier Mitchell and subsequently studied by Charcot, under the title "post-hemiplegic chorea," and now ascertained to be produced by changes in the motor centers on the opposite side. We have further to note that the paralyzed muscles preserve their electric excitability. Under some circumstances the electric excitability may be heightened, under others lessened, but this lowering of electro-contractility becomes more decided the more nearly the paralysis approaches the "spinal" character, which is the case in lesions of the cerebral peduncles, of the pons, and of the medulla. Immediately on the receipt of the injury done by the hæmorrhage, the sensibility is paralyzed with the motion, but the sensibility is soon restored, as a rule, although sometimes the restoration is very gradual, and it is rare for it to be complete. Anæsthesia and analgesia do not accompany lesions of the corpus striatum, whence it happens that these functions are so seldom permanently impaired in hemiplegia. In some cases—lesions of the thalamus, corona radiata, etc.—anæsthesia may be a constant symptom. Anæsthesia may be followed by hyperalgesia, and the paralyzed members may be the seat of neuralgia. Various *trophic* changes occur in hemiplegia. With the first hemiplegia, the paralyzed parts are usually somewhat swollen, are red, and possess a slightly higher temperature, and sweat a good deal. These symptoms subside in a few weeks or two or three months; the affected parts become cold, pale or bluish, the skin scaly and dry, and the nails grow wrinkled, thickened, brittle, and incurved, and the hair changes in texture and length. The skin grows thicker and tougher in many cases, and the larger joints may be the seat of an acute synovitis. In addition to these trophic affections should be mentioned the fact that the paralyzed members in hemiplegia rapidly ulcerate by pressure (bed-sores).

Course, Duration, and Termination.—In the fulminant form death may occur in a few minutes, never less than fifteen. There may be a partial revival, the consciousness restored more or less completely, and then a new attack occurs, closing the scene usually in a day or two. The apoplectic symptoms having disappeared, the next danger consists in the inflammation about the clot, the febrile excitement, headache, and delirium, which usually prove fatal within a week, unless very mild and transitory. Having passed this period there is a partial recovery with hemiplegia, which may gradually disappear, leaving but slight traces of the original mischief. There are but few if any who are restored entirely in all their mental powers, although the motor paralysis may have ceased. If changed in no other way, they are emotional, easily excited to tears, or become altered in disposition, appearing irritable, excitable, peevish. Usually memory is impaired, especially for the events of the time, while matters long past of early life may be vividly recalled. The memory for words may be impaired slightly, may be very defective, or may be entirely lost, constituting the condition of aphasia. This may include inability to express ideas by signs. There may be a gradual decline in the mental powers, the patient lapsing into dementia. The duration of a case of hemiplegia is very uncertain—many continue for ten, fifteen, even twenty years. But hemiplegics are always threatened by a new attack, since the lesions which originally caused it are yet present. Another attack or two is the usual course, proving fatal ultimately unless cut off by an intercurrent disease.

Diagnosis.—As the subject of the distinction between occlusion of the cerebral vessels and cerebral hæmorrhage has been discussed, it remains now to indicate the seat of the lesions by the symptoms. The diagnosis of the position of the hæmorrhage by the symptoms rests on the knowledge of cerebral localizations. Lesions of the cortex and of the medullary substance of the hemispheres give rise to paralysis on the opposite side of the body. If slight in extent, recovery may ensue. A lesion confined to the third left frontal convolution has produced aphasia only. Disturbances in the mental functions are usual and are more decided than the psychical symptoms produced by cerebral hæmorrhage into other parts. Hæmorrhage into the anterior lobe causes paralysis of the opposite half of the body, and aphasia if the left is the seat of the lesion. Hæmorrhage into any of the parts supplied by the left middle cerebral artery will produce disturbance in all the modes of expressing ideas by words and signs. Sensibility as well as motility is disordered in hæmorrhage into the posterior middle lobe and into the posterior lobe. Disturbances of vision and optic neuritis accompany the paralysis, and psychical disorders, with a special tendency to emotional manifestions, are pronounced features. Hæmorrhage breaking into the ventricles is accompanied by formidable symptoms; by deep coma, sometimes by convulsions, partial or general, occasionally by contractions of the paralyzed parts, by unequal pupils, one being widely dilated. Hæmorrhage into the corpus striatum, the most usual site of cerebral hæmorrhage, is followed by paralysis of the members, body, and face on the opposite side; and, if in the left corpus striatum, affections of speech, sometimes complete aphasia, are usually present. There are no disturbances of sensibility in these cases of hemiplegia from hæmorrhage into the corpus striatum. As the optic thalami have never been invaded by hæmorrhage strictly limited to them, the results of lesions are hemiplegia of the opposite side and affections of sensibility. It is probable that the motor symptoms are due to simultaneous injury to the corpus striatum. Hæmorrhage into the pons or medulla is very fatal—in from fifteen minutes to several hours. There are convulsions usually, general muscular resolution, and minutely contracted pupils. If the immediate results are passed over, various motor disturbances ensue: there may be paralysis of both sides, or paraplegia, paralysis of one side, or hemiplegia; paralysis of the members on one side and of the face on the opposite side, or crossed paralysis; also sensory disturbances: there may be anæsthesia with the paralysis of one side, and the paralysis of sensation may be "crossed," as is the motor paralysis.

Treatment.—If the prodromal symptoms threaten an attack of cerebral hæmorrhage, venesection, as the most prompt and efficient means for reducing the intra-cranial blood-pressure, should be at once practiced, the amount drawn being decided by the effect produced. In feeble subjects, leeches to the mastoid may be substituted for venesection. An active purgative (compound extract of colocynth gr. vj, croton-oil gt. j) should be administered. Counter-irritants should be applied to the extremities, and an ice-bag to the scalp. If the hæmorrhage have occurred, these measures will be useless. The utmost quiet should then be maintained, the head elevated, the room darkened. Excellent results are then obtained by the use of tincture of aconite-root, beginning immediately after the coma has passed off. One drop every two hours will usually suffice, as it is not necessary to reduce the pulse by it, unless the reactive fever is considerable, when the dose mentioned may be given every hour for a day or two. When the reaction period has passed, or at the end of two weeks, much may be accomplished by the judicious use of ammonia (ammon. carb. gr. v, liq. ammonii acetat. ℥ ss., four times a day), continuing it for a month or more, or until the retrograde changes in the blood-clot are accomplished. Then the time has arrived for the application of galvanism, a weak current—say from four cups—being passed through the brain in both directions, or from behind forward, and from both mastoids. The application should be daily, and for three minutes at a *séance*. To assist in the restoration, the lactophosphate of lime (sirup) should be administered three times a day with the meals, and the diet should be nourishing and yet unstimulating. As the tendency of paralyzed parts is to waste, the members should from the beginning be subjected to daily massage, at first very lightly, and, if wasting of the muscles is considerable, they should be exercised by faradization. If there is much contraction of the flexors, the extensors should be faradized, and the flexors should receive a continuous mild current to allay their irritability. When there is no longer any local irritation about the site of the hæmorrhage, the injections of strychnine should be practiced into the affected muscles. During the long period after the absorption of the clot, when the paralysis remains stationary or slowly improves, good results are obtained from the persistent use of lactophosphate of lime and cod-liver oil, which act as nutrients to the cerebral matter. These may be given when electricity and the injections of strychnine are practiced.

CEREBRAL HÆMORRHAGE—MENINGEAL.

Pathogeny.—Hæmorrhage into the meninges may be caused by injury; as, for example, the meningeal artery may be ruptured by a fracture, involving the anterior inferior angle of the parietal bone.

The most usual cause, probably, is aneurism, and the vessel most frequently the seat of this disease the basilar, except the meningeal hæmorrhage of newly-born children, which is really traumatic, and produced by forceps delivery. Meningeal hæmorrhage is a complication of the acute infectious diseases. The blood is found in a thin layer, under the dura or in the cavity of the arachnoid, at the base on the hemispheres, and in both situations at the same time. The brain itself may be injured by the escape of blood from an aneurism, and the convolutions may be depressed, the brain-substance pale and exsanguine.

Symptoms.—As meningeal hæmorrhage occurs in the adult, the phenomena attendant on it are the same as those of a large cerebral hæmorrhage. There are coma, complete muscular resolution, often succeeding to convulsions of an epileptiform character, pupils unequal, and reflex movements entirely suspended. Death may occur in a few minutes, or after several hours, in profound coma. In other cases there are headache, dizziness, nausea, and vomiting, drowsiness, passing into stupor, then coma until death after some hours—symptoms supposed to be due to the gradual escape of blood from a ruptured vessel. In new-born children meningeal hæmorrhage is a common cause of asphyxia, from which they can not be roused.

INFLAMMATION OF THE DURA MATER—PACHYMENINGITIS EXTERNA AND INTERNA—HÆMATOMA OF THE DURA MATER.

Definition.—By *pachymeningitis* is meant an inflammation of the dura mater. As this membrane consists of two layers, there are two forms of the inflammation attacking it: *pachymeningitis, externa* and *interna.* Pachymeningitis externa is a surgical malady—an inflammation of the external lamella of the dura, excited by fractures, penetrating wounds, and other injuries of the skull, and by caries of the petrous portion, involving the dura by contiguity of tissue. The last-mentioned malady is so intimately associated with abscess of the brain that it is more appropriately studied in connection with that disease.

Causes.—*Pachymeningitis interna—hæmatoma of the dura.*—Age is an important factor, the tendency to this disease increasing from twenty upward, the largest number *per centum* occurring from seventy to eighty (Huguenin). Three fourths of the cases happen in men, doubtless because they are more exposed to the influences producing this disease. Trauma plays an important part, with or without fracture of the skull. In one of the author's cases the hæmatoma followed a blow on the head—a contusion—with the handle of a heavy riding-whip. No doubt the blow which causes the mischief often is forgotten, and some other cause assigned. A predisposition may be created by several morbid states: by chronic alcoholism, scurvy, pernicious anæmia, Bright's disease, sclerosis of the liver, diseases of the heart, and obstructive maladies of the lungs. Atrophy of the brain, caused by various intra-cranial lesions, seems to be a very important factor in the development of hæmatoma (Huguenin), and to this may be added, by way of illustration, the atrophy of advanced age and of chronic alcoholismus.*

Pathological Anatomy.—The most commonly accepted view is that of Virchow. The first step in the morbid process consists in a hyperæmia of the membrane, and an exudation, developing into a membranous new formation, proceeds from the sub-epithelial layer of the dura.† This neo-membrane contains a multitude of vessels of considerable size, and having very thin walls. Hæmorrhages, often of considerable quantity, take place by the rupture of these vessels, and the size and thickness of the neo-membrane are correspondingly increased. Ultimately the new formation assumes the appearance of a cyst, having a smooth surface exteriorly, and containing within a cavity lined with blood-clot, shaggy masses of fibrin, partly decolorized, hanging from the walls, and a fluid reddish in color and thick with particles of broken-up clot. At a later period there may be no appearances of blood-clot, except, it is probable, some blood-crystals—there may be only a cyst, filled more or less full with a pellucid serum, or instead of a cyst with a single cavity there is a mass of connective tissue, its fibers loosely united, spongy, with serum more or less fully distending the interspaces. Before its nature was understood the cyst containing clear serum was called "cyst of the arachnoid." It should be understood that, between a sac filled with blood-clot and one containing serum only, there are various intermediate grades, the blood being more or less advanced in the process of disintegration, by which all the morphotic elements are dissolved and decolorized. Huguenin ‡ holds that the formation of a hæmatoma is not initiated by an inflammation of the inner lamella of the dura, but that the process consists merely in the organization of a hæmorrhagic extravasation. An immediate vascular communication is established between the dura and the new membrane. The usual position of the new formation is on the upper surface of the hemispheres, extending downward toward the occipital lobe, corresponding to the parietal bone, and in more than half the cases on both sides. The changes in the adjacent portion of the brain are dependent on the size and thickness of the neo-membrane. In a case observed by the author the cyst was a half-inch in thickness at its thickest part, and it depressed the hemisphere correspondingly, the convolutions being flattened, the sulci almost obliterated, and the ventricle lessened one half of its area. Atrophy of the brain, atheromatous degeneration of the vessels, and the alterations in the structure of the brain, accompanying dementia paralytica, are often present. Obstructive diseases of the lungs and valvular affections of the heart are frequently associated with and apparently have a causative relation to this malady.

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* Dr. Jacob Kreminansky, "Ueber die Pachymeningitis interna hæmorrhagica bei Menschen und Hunden," Virchow's "Archiv," Band xlii, S. 129–321.

† Rindfleisch, *op. cit.*, p. 620.

‡ Ziemssen's "Cyclopædia," vol. xii.

Symptoms.—There is necessarily much obscurity about this disease, and the symptoms are diffused, and but little characteristic. There occur first the indications of excitement of function, followed by those of depression. In the first group are an obstinate headache, vertigo, singing in the ears, contraction of the pupils to a marked extent, uncertainty and feebleness in the movements, without paralysis, wakefulness, and when sleep comes it is disturbed by exciting dreams. In some cases, but less frequently, there occurs an attack, apoplectic in character and with the usual phenomena of that state. The period of excitation continues from a few days to three months, and is succeeded by the signs of cerebral depression. At this point in these cases there will usually occur attacks like those of cerebral hæmorrhage and from the same cause, but in this stage of this disease they are apt to pass slowly into unconsciousness. Death may occur in this coma, or the patient emerges from it slowly, when there will appear the symptoms due to the hæmatoma now produced. It should be remembered that this new formation is on the surface of the hemisphere, that there has been no destruction of the cerebral tissue as in cerebral hæmorrhage, and that compression is exerted by it on the brain-mass on one or both hemispheres. The symptoms now present are persistent headache, contracted pupils, and paroxysmal attacks of somnolence, persisting for days at a time. If the pressure is on one side only, the corresponding pupil is smaller. Paresis of the muscles, contractions, twitching of the muscles, are observed on one side when the lesion is unilateral, or they may be double. Convulsive movements, limited to a hand, or arm, or leg, may be observed. Hemiplegia may slowly develop out of a unilateral paralysis. After existing on one side for a time, these motor disturbances may slowly affect the other side, doubtless because of an extension of the disease. In one third of the cases there are defects or embarrassment of speech, but rarely complete aphasia. There are not any disorders of sensation. The pulse is usually weak, rapid, and rather irregular. Fever has been noted in many cases. The pulse may be slow during the hæmorrhage.

Course, Duration, and Termination.—The first stage, or that of excitation, usually lasts but a day or two, yet in exceptional cases it may continue a month or two. Death may occur in the apoplexy. The period of depression lasts usually from a week to one month, and may continue a year, but the most common duration is about twenty days. Although death is the usual result, recovery may take place, but it is doubtful whether the mental faculties are ever again entirely restored.

Treatment.—The remedial management of this disease is a discouraging undertaking. The usual remedies for cerebral hyperæmia may be used for the symptoms of excitation.

ACUTE HYDROCEPHALUS—INFLAMMATION OF THE PIA MATER: LEPTOMENINGITIS.

Definition.—The term *hydrocephalus* signifies water in the brain, but is restricted to a disease characterized by the presence of a serous fluid in the arachnoid spaces, in the pia mater, in the brain-substance (œdema), and in the ventricles. Hydrocephalus may be congenital or acquired. In this form—the inflammatory—the presence of water is due to an acute inflammation of the *pia mater*, but without tubercle—leptomeningitis. Hydrocephalus is a general term which serves to unite various conditions, but it is always a symptom.

Causes.—Mechanical causes, which prevent the return of blood from the vena Galeni and the right sinus, will induce effusion into the ventricle. Intra-cranial tumors, bands of false membrane, obstruction of a sinus or tumors of the neck so situated as to compress the jugular vein, belong to this category. Disease of the right heart, obstructive diseases of the lungs, as emphysema, sclerosis, etc., may cause hydrocephalus by mechanical interference with the circulation. In advanced age, ventricular dropsy occurs in consequence of atrophy and shrinking of the brain. Various cachexiæ affect the intra-cranial circulation and cause dropsy, as Bright's disease, cancer, tuberculosis, etc., but only the first-named stands in a causative relation to the form of hydrocephalus here considered. Dropsy of the ventricles coincides with general dropsy from cardiac and renal diseases. Leptomeningitis is a disease of early life, from one to five years of age, but it may occur at any age. Unfavorable hygienic conditions increase the tendency to it, and the predominance of the nervous system in the bodily conformation invites this, as other forms of nervous disease. Both sexes are affected alike. Among the exciting causes may be mentioned dentition, the eruptive fevers, and blows on the head.

Pathological Anatomy.—The effusion is usually confined to the ventricles, but there may be considerable distention of the subarachnoid spaces, œdema of the pia and of the neighboring portions of the brain. When the effusion is limited to the ventricles, the brain-tissue is found to be moister from the gray matter inward. More or less softening by imbibition exists for a short distance from the ventricles. The choroid plexus is hyperæmic, and may contain minute extravasations. The ventricles are usually symmetrically dilated, but, in the hydrocephalus of the aged, one ventricle may be very much dilated and the other encroached on and narrowed.

Symptoms.—There are several modes of onset, and several types of cases, as the causes sufficiently indicate. One variety, known as "serous apoplexy" by the older writers, begins, by reason of a sudden effusion, very abruptly, with the phenomena of apoplexy: there are unconsciousness, muscular resolution, immobile pupils, involuntary evacuations. In the midst of the coma there may sometimes arise delirium. So extreme may be the pressure of the fluid that the medulla oblongata ceases to functionate, and the patient dies in a few hours, and rarely is life prolonged several days. The next type may be characterized as the *convulsive.* This begins with the symptoms of excitation, and there may be some feverishness, headache, nausea, and vomiting, for a few days, when an attack of eclampsia occurs, or the convulsion may be the initial symptom, or in adults a violent delirium. These symptoms are soon followed by depression, and the patient passes into a stupid, somnolent state, is roused with difficulty, and weakness of the members is succeeded by complete paralysis. Very unexpectedly, sometimes, the consciousness revives, but for a brief period, and the coma comes on again, death soon occurring. This form usually appears in the course of Bright's disease or general dropsy. The ordinary form in children sets in with feverishness, headache, intolerance of light, and corrugation of the forehead; intolerance of sounds, restlessness, delirium toward evening, wakefulness, or disturbed sleep; vertigo, twitching and spasmodic contraction of muscles (head drawn back, fingers and toes incurved); great sensitiveness of the skin, pain being caused by a slight touch, especially about the neck; nausea and vomiting without cause, the belly drawn in, and obstinate constipation. Such symptoms will continue for several days, when there will occur convulsions of an epileptiform character, or partial convulsive movements in an extremity, in the muscles of the abdomen, or in the face. The temperature may rise very high during these convulsive attacks—the pulse rapid, and often irregular—but the temperature declines after the eclampsia has ended. Death may take place at this period, or, as is most usual, the epileptiform attacks cease and the ordinary course of the disease is resumed. The symptoms of depression now come on: restlessness is replaced by stupor, rigidity and contraction of the muscles by paresis, heightened sensibility by anæsthesia. The pupils dilate somewhat and become less and less mobile, and are often unequal in size, and double vision is noticed. The pulse declines in force, and exhibits a marked degree of inequality, now beating at 80, now at 130. The respirations become irregular in rhythm, and manifest the Cheyne-Stokes type to some extent. The surface becomes cool; the fontanelles are prominent and rounded; and the sutures in young infants separate somewhat. The vomiting continues, and the nutrition is greatly impaired. The patient sinks into a deep coma, and, although there occur remissions, in which the unconsciousness seems less profound, the pulse and breathing better, and the reflex movements more easily excited, they do not persist.

Course, Duration, and Termination.—A few cases have been reported cured. They were milder examples of the common type, as seen in children, and, although the symptoms of excitation were well marked, those of depression did not come on. The appearances of improvement, which are observed in the stage of depression, are illusory. The apoplectic and convulsive forms are always fatal in a few hours or two or three days; the common form very rarely terminates in recovery. The duration of the cases terminating by exhaustion is very protracted, reaching to four, six, even eight weeks, but the average duration of these cases is about three weeks. Those ending by convulsions do not often continue beyond two weeks. The extended duration of some cases is due to the absence of convulsions and the prolongation of the stage of coma. As the questions connected with diagnosis and treatment are the same as for tubercular meningitis and for simple meningitis, they are postponed for separate and full consideration at the conclusion of the subject of meningitis.

CHRONIC HYDROCEPHALUS.

Pathogeny and Symptoms.—*Chronic hydrocephalus*, as it occurs in children, usually succeeds to the acute form, and is a result of rickets, or an accident of the rachitic constitution. The quantity of fluid is much greater, however, in the acute form. After youth, the accumulation of fluid is due to the pressure of tumors on the straight sinus, vein of Galen, etc., and in old age considerable effusion is produced by atrophy of the brain. In dementia paralytica, there may be considerable distention of the ventricles and of the perivascular lymph-spaces. The initial symptoms are those of irritation, and are due to the presence of the new vascular conditions, but, as the effusion grows, the neighboring parts are pressed upon, and the symptoms of depression then dominate the situation. Hebetude of mind, stupidity, diminished activity of the special senses, and a fatuous expression of countenance, are now observed. General sensibility—tactile, heat, cold, and sensory—is much less active than normal. Motility is also impaired, especially in the distribution of the seventh nerve: there are present ptosis and a blank expression due to relaxation of the muscles of expression. The pupils are unequal, and respond sluggishly to the action of light. The tongue is paretic, and the speech thick and utterly unintelligible. The faculties continuously decline into idiocy or dementia; locomotion becomes impossible; control of the sphincters is lost; sight and hearing are abolished. This slow decline may be diversified by convulsive seizures, or more acute symptoms may be produced by a sudden and large effusion. In the latter, unconsciousness may occur, preceded by violent headache, and followed by inequality of pupils, hemiplegia more or less complete, slow, irregular pulse, impaired articulation, aphasia, etc. The duration of the cases is measured by months, and the termination is fatal. The fatal result may be caused by the ordinary progress of the disease—the compression of the increasing effusion, or by some intercurrent disease, as pneumonia, pleuritis, meningitis, etc. The treatment is the same as for the congenital form, to which the reader is referred.

CONGENITAL HYDROCEPHALUS.

Causes.—Much obscurity obtains on this point. Imperfect formation of the cranium and defective development of the brain are influential causes. A chronic inflammation of the ependyma seems to develop the disease sometimes. Again, it is the product of purely mechanical agencies, such as the compression, by a tumor, of the straight sinus or of the vena Galeni.

Pathological Anatomy.—There is no constant ratio between the size of the head and the amount of liquid present. The fluid may vary from an ounce or two to sixteen ounces or more. The liquid is transparent, of a straw-color, and contains but little solid matter, which consists of albumen and chloride of sodium. If the fluid is considerable, the ventricles are much distended, the optic thalami and the corpora striata are depressed and flattened, the orifice between the two ventricles is very large, and the roof of the ventricles is thinned according to the amount of fluid, and may be to the extent that only a mere line of white and gray matter remains. From this extreme distention to the mere filling of the ventricles without disturbing the harmony and proportion of parts, there are numerous variations in the quantity of fluid. The enlargement of the head caused by the effusion may be sufficient before birth to impede or prevent natural delivery. The degree of ossification is an important element in the dimensions. The bones are so thinned as to be translucent ; the fontanelles and the spaces between the sutures are very wide ; the lateral portions of the cranium project greatly ; the forehead bulges out enormously over the eyes ; the orbital plates are depressed, whence the eyes are forced forward between the lids, producing the condition of exophthalmus.

Symptoms.—The dimensions of the head at first attract attention to the condition of the infant. At the period when the head should be held erect it is found to droop, resting on one or the other shoulder. Then it is noticed that the mental development does not grow with the physical ; that the face is devoid of expression ; that the attention is not attracted by surrounding objects ; that voluntary movements are slow of execution. When the period for standing on the feet and making attempts at walking arrives, the power to maintain the erect posture is wanting. The general condition is not favorable, and, although the appetite may be voracious, the assimilation is not equal to the preparation of the aliment for absorption. The face has a rather old look, and is wrinkled; the voice is feeble and sibilant. Some of these subjects are, however, capable of slight mental development, but they do not acquire any higher capacity for speech than the automatic use of a few words, and, if they reach manhood, the mental powers are only those of a child, the voice having the same characteristics. As regards the special senses, odor and taste are more often preserved, while hearing is imperfect. Disorders of vision and of the cutaneous sensibility are common. Numbness, tingling, and pains are felt in the extremities. Motility is impaired to a less or greater extent. There may be a general paresis, which is more pronounced in one member, but rarely complete paralysis. There are great differences in the cases: some can not stand without support; others walk, but the gait is hesitating; they stumble at every obstacle, and seem constantly to be about to pitch forward, owing to the weight of the head. Epileptiform attacks occur in many of the cases from time to time. The nutrition is bad, notwithstanding a voracious appetite; they suffer from constipation, and have an excessive flow of saliva from the mouth; the skin is dry and the eyelids are puffy. If the anterior fontanelle is very large, strong compression will put the patient into a somnolent, even a comatose state.

Course, Duration, and Termination.—The course of the disease is chronic, its march irregular. At times considerable progress is made; then the case remains stationary for some time, even for years. A majority of the cases terminate within the first year; others are prolonged to the fifteenth year, even beyond this. The more voluminous the head, the more rapid the progress of the case, as a rule. Spontaneous cures have been effected by the discharge of the liquid, either by a wound or through the nose. Cures may be effected in slight cases when recognized early, but such a result is exceptional, the usual termination being death. The fatal result is reached by convulsions and coma, unless previously by some intercurrent diseases.

Treatment.—The author has had good results from the use of iodide of potassium, but it was a case of effusion probably, limited to the ventricles. Flying-blisters, the internal administration of digitalis, ergot, and purgatives, with the occasional use of iodide of potassium, carried to slight iodism, are the remedies best adapted to the cases of slight extent, which may be conducted to a favorable termination. The use of the finest aspirator-needle may now be justified, in view of the spontaneous cures which have followed accidental discharge of the fluid. Care being taken to avoid the longitudinal sinus, the ventricle may be entered with safety, and the operation is easily performed. When sufficient fluid is withdrawn, the cranium should be gently but firmly compressed.

TUBERCULAR MENINGITIS.

Definition.—By this term is meant an inflammation of the cerebral meninges, caused by the presence of tubercular granulations.

Causes.—Tubercular meningitis occurs most frequently in children from two to six years of age, and in adults from twenty to thirty years, and is about equally distributed between the sexes. Children of the well-to-do classes are apt to suffer from this disease, and those whose nervous system preponderates over the digestive and muscular. The "gelatinous children of albuminous parents," as the phrase goes, possess a special susceptibility to tubercular meningitis—the pale, thin-skinned, blue-eyed, precocious children of pale, flabby, and delicate parents. The changeable weather of winter and spring disposes to the development of the disease. All the circumstances comprehended in the term *bad hygiene* promote the occurrence of this malady, especially insufficient light, bad air, and improper food. None of these causes could produce this disease in the absence of the tubercular matter. It is extremely rare to find the tubercular deposits limited to the pia mater—in thirty-eight examples of the disease there were but two in which the deposit was thus limited (Jaccoud). Tubercular meningitis is transmitted by inheritance in the limited sense that the diathesis is inherited: in one member of a family so tainted it may be meningitis, in another phthisis, in a third ulceration of the intestine.

Pathological Anatomy.—Miliary tubercles, in the form of grayish-white granules having a translucent and somewhat gelatinous appearance, are distributed along the vessels of the pia mater. These miliary granules vary in size from a minute object just visible to the eye up to a large pin's-head, and these aggregating in a mass form a tubercle as big as a pea. The distribution of the tubercle-granules is not the same in all situations: it may be greater in the neighborhood of the arteries of the base (basal meningitis) or the arteries of the convexity; again, the principal deposits may be in the pia of the frontal or of the parietal regions. There may be but few tubercles in any situation in some cases; in others the whole membrane may be thickly studded with them. The intensity of the inflammation does not have a constant relation to the number of tubercles, for the inflammation may be great with few tubercles, and slight with a large crop of tubercles. Besides tubercle there are present the evidences of suppuration in a sero-purulent effusion, seen along the course of the vessels especially, as "yellowish stripes" (Rindfleisch). The pia mater at the base is thickly covered with a gelatinous exudation, and the membrane itself is thickened and opaque, especially about the optic chiasm and the anterior perforated space extending up into the fissure of Sylvius. There is more or less effusion usually in the ventricles, and the plexus choroides is the seat of an extreme hyperæmia. More or less œdema of the cortex takes place, provided there is no effusion, but when there is effusion the cerebral substance is dry and anæmic from pressure. Miliary tubercles are also found in the cortex, and migrated white corpuscles are abundantly distributed through the cerebral tissues. The miliary tubercles, aggregated in masses, are found in many situations to have undergone caseous or fatty transformation. Tubercles are also widely distributed throughout the body.

Symptoms.—There is a period during which it is probable tubercular deposit is taking place, manifested by symptoms which may be justly called prodromal. The disturbances resulting in the symptoms of the disease are produced by the inflammation which is excited by the tubercular deposit. The prodromal symptoms are chiefly those indicative of failure of nutrition ; emaciation goes on, and the strength declines proportionally ; the appetite fails, and the character changes, the patient becoming irritable and morose. The child, before precocious and vivacious, becomes indifferent to former occupations and amusements. Sleep is disturbed by vivid dreams ; the child grinds its teeth, cries out suddenly in the night, and walks about in a somnambulistic state. The digestive organs become disordered, the belly is swollen, diarrhœa alternates with constipation, and vomiting occurs without cause, without the presence of indigestible matters to excite it. Headache is complained of, vertigo is experienced in rising up to walk or in lying down, and pains are felt in the limbs. The ominous symptom of double vision is sometimes observed at this period. The author has heard a precocious little boy say during this prodromal period, "I see two mammas," several weeks before the developed disease came on. The stage of excitation symptoms appears in from two days to six weeks, even longer, of the prodromal period. Fever begins ; the temperature rises to 102° or 103° Fahr. in the evening, and falls in the morning to 99° ; the pulse varies greatly, going up to 130, 140, and falling to 80. In adults this fever of the excitation period may be wanting. At all times during the disease the pulse is very unequal in rhythm and the heart very excitable. The pulse may become slow and regular without any apparent reason, or may again become very rapid. Although the type of the fever is remittent and is often mistaken for remittent fever, it is subject to great variations. Three important symptoms besides the fever mark the onset of the excitation period—headache, vomiting, and constipation. The headache is severe, heavy, or lancinating ; and, although continuous, is varied by exacerbations, compelling outcries, or rubbing the head, or other manifestations of severe suffering. As the suffering is increased by light, the head is either buried in the bedclothes or turned to the wall, or the eyes are covered by the eyelids. The vomiting occurs a few times during the twenty-four hours, and is always without apparent cause ; the constipation persists obstinately ; the belly is hard and retracted. During the excitation period, changes in the character and disposition which began in the prodromal period continue and are more pronounced—an exceeding fretfulness and hostility to those to whom they were much attached, developing. Not only the special but general sensibility is exalted; all movements cause pain and loud expressions of suffering, and the least pinch, especially about the neck, excites exquisite pain. In the motor sphere the symptoms of excitation take the form of spasmodic movements of muscles, contractions, and rigidity, especially seen in the muscles of the members and of the neck. There will occur at this period also local convulsive movements, and not unfrequently general convulsions (eclampsia), with the usual phenomena. The stage of excitation due to the development of meningitis now begins to yield to the phenomena of depression due to the pressure of the fluid on the cerebral matter. Here, then, is a period during which the symptoms of irritation still linger, and the symptoms of depression are just manifesting themselves—*a mixed stage:* paroxysms of pain and spasmodic and convulsive attacks are separated by periods of somnolence, during which there may be uttered the peculiar shrill, unearthly cry or shriek called the *hydrocephalic cry.* If attempts at walking are now made, the patient's movements are incoördinate and uncertain, and indeed it is impossible to preserve the equilibrium. Torpor now becomes the settled state, but still the patient can be roused to make an imperfect or monosyllabic reply to questions, lapsing back into a somnolent state as soon as the attention is no longer attracted. At this period the ocular changes are manifest: there are strabismus and double vision; the pupils are often unequal. The countenance is pale, stolid, and expressionless. The retinal changes are very pronounced. Tubercles of the choroid can often be detected. At first the optic papillæ are swollen, blurred, and indistinct, the veins are enlarged and tortuous; but in the further progress of the case retrograde changes, ending in white atrophy of the disks, take place.* This mixed stage has a variable duration of a few days to a week or more, and is varied by illusory evidences of improvement, which often mislead the physician, and raise false hopes in the minds of the parents and friends. These appearances of improvement at this time consist in a more regular pulse, less somnolence, greater interest and attention to surrounding objects, playthings, etc. Indeed, it seems as if the morbid process were arrested, and that convalescence is about to be established; but, while the most cheerful anticipations are indulged in, formidable symptoms suddenly appear. A general convulsion, it may be, occurs, or the muscles of the neck and spine become rigid, or local convulsions affect the members; a mild delirium manifests itself; the respiratory movements become very unequal in depth and irregular in rhythm, and have at times a sighing character; the pulse is equally irregular, becomes slow, falling to fifty even, and there are marked variations in its volume and tension; the temperature remains elevated, but preserves its remittent type. The approaching stage of depression is now announced by the increasing somnolence, by the greater effort to excite the most transient and indefinite response; light nor sounds no longer disturb the brain; sensibility is no longer excitable; the contractions of muscles are replaced by relaxation; the urine is passed involuntarily. When the stage of depression is fully established, no indication of consciousness can be excited by any irritation, and the reflex movements of the eye are entirely abolished. The pupils now dilate; the upper lids droop over the eyes; the globe of the eye rolls from side to side (nystagmus); the pharynx becomes less and less responsive to the presence of food or drink, and finally no movements can be excited—only the slow, irregular pulse changing to a rapid and feeble pulse and the Cheyne-Stokes breathing manifest the signs of functional action. *Râles* from accumulating mucus now obstruct the breathing, the pulse becomes more rapid and feeble, a cold sweat breaks out on the skin, the abdomen becomes full and prominent, the evacuations are relaxed and involuntary, and death occurs at last by protracted failure of respiration or by a convulsion.

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* Allbutt on "The Ophthalmoscope," p. 112.

Course, Duration, and Termination.—The division into periods is an arbitrary arrangement, but useful as a means of indicating the variability of the symptoms and their relation to the morbid process. But the course of the disease is not always that above indicated: there are variations due to the age of the subject; and tubercular meningitis, as a secondary disease, differs from the primary affection. In acute tuberculosis the cerebral symptoms are pronounced, but they are not those of tubercular meningitis. The form of the disease occurring in adults is secondary, usually to advanced pulmonary tuberculosis. There are no prodromal symptoms. In the midst of a pulmonary disease, the patient experiences intense headache, vertigo, delirium, often of a maniacal character; there occur contractions of muscles, followed by paresis; irregularity of pulse and respiration is noted; and coma and insensibility succeed to wakefulness and delirium. Convulsions do not occur in the course of this secondary meningitis in adults.

The prodromal period in the ordinary form of the disease has no fixed duration, and may continue for three months; it is usually about three weeks, and is probably never absent if carefully inquired into. The period of excitation has a duration of about one week to two weeks; the middle period may be protracted three weeks, but usually occupies one week; the period of depression lasts from one to two weeks. Although a very few cases have been reported cured, it is held to be an incurable disease, and the termination fatal. The cases reported cured were, it is generally supposed, examples of simple not tubercular meningitis. The consideration of diagnosis and treatment will be taken up after the study of simple meningitis of the base and convexity.

ACUTE MENINGITIS.

Definition.—*Acute meningitis* consists in an inflammation of the pia mater and arachnoid, chiefly the former. It may be limited to the base—*basilar meningitis*, or to the convexity—*meningitis of the convexity.*

Causes.—Meningitis is derived by contiguity of tissue from disease in neighboring parts—disease of the internal ear, erysipelas of the face, malignant pustule, caries of the bones, traumatic injuries. It is then entitled *secondary meningitis.* It sometimes arises during the course of inflammation of serous membranes, acute rheumatism, puerperal fever, pyæmia, Bright's disease, by that which was formerly called a metastasis, and hence was designated *metastatic meningitis.* The primary form with which we are now chiefly concerned arises from the causes inducing congestion and overaction of the brain, as excessive intellectual effort, prolonged wakefulness, exposure to the direct rays of the sun, and alcoholic excess. The most common cause of meningitis is the deposit of tubercle, but this has been discussed in the previous chapter. The primary form is a rather uncommon malady. The disease is more frequent in men than in women, and is less common in children.

Pathological Anatomy.—In the basilar form, the inflammatory changes are confined to the base, and consist of intense hyperæmia, followed by purulent and fibrinous exudation, covering the parts at the base as far back as the pons, and forward to the optic chiasm, and surrounding some of the nerves. The choroid plexus is intensely hyperæmic, and the ventricles may be distended with fluid, compressing the hemispheres and flattening the convolutions. The ependyma of the ventricles becomes granular or undergoes thickening. Hydrocephalus is by no means present in all cases. In the meningitis of pyæmia and other septic maladies the fluid exuded is largely purulent, and migrating white corpuscles are found in great numbers in the exudation in the ventricles. In meningitis of the convexity the inflammation is excited by extension from the bones of the cranium, from caries of the petrous portion, from panophthalmitis, from erysipelas of the head, and carbuncle of the upper lip, etc., and is of the character manifested by the same process at the base. Pus is extensively infiltrated, especially along the course of the great vessels. The migrating white corpuscles invade the gray matter of the cortex, and pus-cells are contained in the fluid of the ventricles in large numbers. Although the morbid process may be confined to the convexity, yet in most cases the base is more or less invaded.

Symptoms —There may or may not be a prodromal period, characterized by a rather violent headache, vertigo, and cerebral vomiting, lasting for a few hours or a day or two. The real onset of the disease is rather sudden, and, like other acute inflammatory diseases, begins with a decided chill followed by high fever—by a more intense and sustained fever than in other cerebral maladies. The pulse may be 100, the temperature 103° or 104° Fahr. The face is flushed, the eyes are injected and swollen. There are from the beginning an intense headache, vertigo, nausea, and vomiting. When the morbid process is confined to the base, the mental symptoms may be very insignificant, and consist of confusion of mind, or mild delirium toward evening or on awaking from sleep, but usually there are hallucinations and illusions, active delirium, sometimes furious and maniacal, and these are proper to meningitis of the convexity. During the period of excitation there are hyperæsthesia of the skin and contractions and spasms of the muscles of the extremities, and those innervated by the cranial nerves—hence the ocular defects and disturbances, twitchings of the facial muscles, rigidity and contraction of the spinal and cervical muscles, etc. The symptoms of excitation are soon succeeded by depression. Early, besides the muscular incoördination and consequent ataxic aphasia, there occurs a true aphasia from deposits along the middle cerebral and consequent compression of the supposed language center. Delirium is succeeded by somnolence, gradually deepening into coma; exalted sensibility (hyperæsthesia) yields to loss of the senses of touch and pain; spasms and contractions of muscles are replaced by paralysis. The pupil dilates. Early in the disease ophthalmoscopic examination discloses choked disks and swollen veins, but the papillæ rapidly undergo atrophy. The eyelids drop down upon the eyes, and are swollen and prominent; epistaxis often occurs. With the increasing pressure on the medulla oblongata, the pulse falls, then grows rapid and feeble, but the temperature continues at 103° or 104° Fahr. The respiration becomes irregular, sighing—of the Cheyne-Stokes type—and increasingly shallow.

Course, Duration, and Termination.—The cases of meningitis present great variability in their course and in their duration: some are characterized by remissions—apparent improvement continuing for days, and followed by relapses. Again, the course and duration of other cases are much affected by the cause of the meningitis and the character of the coexistent malady. The duration may be stated as varying from one week to eight weeks. The usual termination is in death. Cures may be effected in which permanent damage has happened, and a sense or a member remains only partly capable of function ever after. Perfect cures have been reported, but a doubt of their genuineness must always be entertained. Before and immediately succeeding death the temperature may rise to 105° and 106° Fahr.

Diagnosis.—This question includes the differentiation of the several forms of meningitis, and the separation of meningitis from acute tuberculosis, typhoid fever, tumor and abscess of the brain, encephalitis, cerebral hyperæmia, uræmia, and disease of the labyrinth. Tubercular meningitis is differentiated from the other forms by the history, by the simultaneous appearance of tubercular deposit in other organs, especially pulmonary tuberculosis, and by the presence of tubercles in the choroid. Acute hydrocephalus is distinguished from meningitis by the less degree of fever, by the predominance of the stage of depression, and, in the apoplectic and convulsive forms, by its more speedy termination, and by the absence of symptoms due to the implication of the cranial nerves at the base. Meningitis in its various forms is distinguished from acute tuberculosis and typhoid fever by the symptoms of excitation of the brain, especially the convulsions, and subsequently by the ocular and other paralyses, the alterations of the retina, by the absence of the rose-spots, the absence of diarrhœa, and the presence of constipation. Meningitis is distinguished from tumors of the brain by its more rapid progress, more diffused symptoms, and the presence of fever; from abscess, by the absence of a period of latency after the symptoms of an inflammation; and by the diffusion of the symptoms of depression. From cerebral hyperæmia, meningitis is differentiated by the higher temperature, longer duration, and the symptoms of depression succeeding to a stage of excitation. In uræmia the temperature is usually below rather than above normal; the urine is scanty and contains albumen, and there is or has been dropsy. Labyrinthine disease, even inflammation of the middle ear, may closely simulate meningitis, but the existence of ear-symptoms and the absence of paralysis indicate the source of the symptoms, which also begin with great violence.

Treatment.—The head should be kept elevated; the room dark and quiet, to exclude all sources of cerebral excitement. An ice-bag should be put to the head, the hair being previously removed. If a robust subject, leeches should be applied to the mastoid bone and to the nape of the neck. An active purgative should be administered at the outset. If the temperature is high, the wet-sheet should be used two or three times a day, unless mental excitement is produced by it. If the patient is calm under its use, and if the temperature is lowered by it, the best results may be expected from it. The author has witnessed admirable results from the administration of the tincture of aconite-root (two drops) and the deodorized tincture of opium (five drops) every two hours during the stage of excitation. Bromide of potassium (ʒ ss.) and fluid extract of ergot (ʒ ss.), every four hours, are appropriate remedies to diminish the vascular excitement, but, in the author's experience, are not so successful as aconite and opium. If there be much cerebral excitement, good results are obtained from the fluid extract of gelsemium, which may be added to the other remedies (♏j every two hours). If the convulsions are numerous, bromide of potassium must be administered freely, and chloral given by the rectum. During the whole duration of the disease up to coma, Lugol's solution (four to ten drops *ter in die*) should be administered, or the iodide of potassium if better borne. This remedy is especially serviceable in the tubercular form. During the stage of excitation, mustard-plasters should be applied to the forehead and neck several times a day, allowing them to remain on but a minute, or even less, until slight rubefaction is produced. The author must decidedly condemn the practice of severe and protracted counter-irritation so often pursued in cerebral maladies. The remedies above advised must be discontinued when depression of function occurs—except the iodine solution or iodide of potassium. The best results are then obtained by small doses of quinine, with belladonna tincture or extract (two grains of quinine and one sixth grain of belladonna extract every three hours). An occasional or spasmodic administration of these remedies will not suffice—they must be persisted in. During this period careful alimentation is very necessary, and wine may be sometimes very serviceable, but its administration must be watched. The author feels it his duty to condemn the use of mercury in this disease. Experience has shown that it has no power to check the inflammation, and ptyalism enhances all the dangers.

CHRONIC MENINGITIS.

Pathogeny.—*Chronic meningitis* is characterized by the formation of membranous exudation, opacities of the arachnoid, adhesions between the arachnoid and pia, and such firm attachment of the membranes to the brain that, in detaching them, the brain is torn. The morbid changes in the membranes, the formation of neo-membrane, etc., take place both at the convexity and at the base. In the latter situation the cranial nerves are impinged on with the effect, first, of causing irritation, shown in pain and spasm of these nerves at their peripheral distribution; and, second, loss or depression in function, exhibited in anæsthesia and motor paralysis. The lesions of chronic meningitis are found in old cases of mania, dementia, and dementia paralytica. The only causes known to have an effect in producing this disease are injuries of the head, chronic alcoholism, and heredity.

Symptoms.—So often associated with the mental disorders above mentioned, chronic meningitis is obscured and overlooked in the more pronounced symptoms of the associated malady. There are, necessarily, two classes of symptoms to be noted—those of irritation, those of depression: the former mean pain, spasm, or contraction; the latter anæsthesia and paralysis. In the first group are headache, *tinnitus aurium*, vertigo, double vision, rigidity and contraction of the muscles of the neck and spine, nausea and vomiting, irregular pulse, and a rhythmic breathing ; in the second, impaired mind, defects of speech, or aphasia, amaurosis (double optic neuritis), weakness, paresis, or paralysis of members or of groups of muscles, weak pulse, and sighing, shallow, irregular breathing, paralysis of tongue, and paresis of pharynx, etc. The treatment is that of the acute form, except the use of the arterial sedatives.

ACUTE ENCEPHALITIS—ABSCESS OF THE BRAIN.

Definition.—By *acute encephalitis* is meant a suppurative inflammation of the brain, and which is localized, not diffused. It may be *primary* or *secondary*.

Causes.—Notwithstanding certain stimuli, long acting, have been supposed to cause inflammation of the brain, the facts do not warrant this supposition. These supposed causes are, prolonged mental effort, exposure to the sun's rays, venereal excesses, alcoholism, etc. The affection is more common in men than in women (nine to four), and occurs at all ages, but especially at the most active period in life—from puberty to fifty years of age. The secondary is probably the only form of the disease, and arises from injury and contusions of the head ; disease of the nasal fossæ, frontal sinuses and orbit ; caries of the cranial bones, and especially of the petrous bone, from disease of middle ear. Besides traumatism, the most frequent cause is caries of the bones. Rarely encephalitis has occurred in the course of acute infectious diseases, and more frequently from infective emboli.

Pathological Anatomy.—The points of inflammation are always circumscribed, and vary in size from an almond to an orange. They may be multiple, or occupy several parts at the same time, but this is not usual, and when so the individual collections are small. The usual position of the inflammation is in the corpora striata, optic thalami, the gray matter of the cortex, the cerebellum, the abscess forming in the white matter of the hemispheres. They are said to be more frequent in the left than in the right hemisphere. The abscesses may or may not be, but usually are encysted, or inclosed in a limiting membrane. They are irregularly circular in shape, and when not encysted the walls of the cavity are extremely irregular, masses of breaking-down cerebral matter projecting into the pus, which is also diffused into the surrounding textures. The abscess is composed of rather thick, greenish, odorless, but sometimes fetid pus and disintegrated remains of the cerebral tissue. The initial change at the site of the abscess is hyperæmia ; minute extravasations take place (capillary hæmorrhages), giving to the inflamed area a dark, reddish color, whence the term *red softening ;* migration of white corpuscles, diapedesis of some red corpuscles, and exudation of serum holding albumen and fibrin in solution, occur simultaneously. The brain-tissue, being soft and easily broken up, is rapidly disassociated, and its elements disintegrated, and in a short time a soft, pultaceous red mass results, which more and more assumes a purulent character, becoming first reddish-yellow, then yellow or greenish-yellow, ultimately almost white. The limiting membrane consists of a connective-tissue material constructed from the neuroglia. The part which the cells of the neuroglia and the cellular elements of the gray matter (which most readily takes on the suppurative inflammation) assume in the process is not definitely known, as Rindfleisch frankly admits. The encysted abscess may take either of two directions: the pus may be gradually absorbed, the cyst undergoing calcification, or, after a quiescent period, set up a new disturbance, ending in death, which is vastly more common. When the abscess approaches the surface, meningitis is excited and adhesions of the membranes may take place to neighboring parts and to the walls of the abscess. The injury caused by an abscess is not limited to the portion of brain inflamed, but the neighboring territory is in the condition of collateral hyperæmia and œdema.

Symptoms.—There are three stages in the course of encephalitis: inflammatory; period of silence; coma. Not all conform to this, and hence variations must receive some attention, and the symptoms are much influenced by the locality of the lesions. There are symptoms common to cerebral abscess, and symptoms only produced by abscess in certain situations. The symptoms of the inflammatory stage are headache, vertigo, noises in the ears, double vision, strabismus (temporary), sometimes affections of speech, numbness and tingling in certain members, sudden muscular cramps, incoördination of muscles in walking, sometimes nausea and vomiting without cause, irritability of the bladder, etc. If these symptoms have followed a blow on the head, or have come on in the course of an otorrhœa, or of a long-standing affection of the nose, attention should be directed to the probable development of an encephalitis. After some days or weeks of these symptoms an apoplectic seizure may occur, or convulsions of an epileptiform character or delirium. Rigidity and contraction of one side or of both sides are found to exist, succeeding the seizure, the period of unconsciousness being short; also strabismus, double vision, and embarrassment of speech (amnesic aphasia). Sometimes the members contracted, sometimes on the other side, are attacked by clonic spasms, and occasionally there are general convulsions of an epileptiform type. The intellect is not always disturbed at the beginning, but there may be acute maniacal delirium or simply confusion of mind. It rarely happens that paralysis —a symptom of depression—appears as an initial symptom, and, if so, it may be safely assumed that the symptoms of irritation escaped notice. Heightened general sensibility—hyperæsthesia—is present in the parts, the seat of contractions or spasms, but anæsthesia accompanies the period of depression. These symptoms of the inflammatory stage are attended by fever, not of a special type, the thermometer rising to 102° or 103° Fahr. The pulse is at this period full and strong. The urine is scanty and high-colored. Nausea and vomiting are very persistent symptoms in some cases, and occur to a greater or less extent in all, and this statement is equally true of constipation. The inflammation stage proceeds to the formation of pus, and includes the incapsulation of the abscess. When the purulent elements are diffusing through and disassociating the nervous tissue, the symptoms of depression succeed to excitation. The formation of pus may take place in five or six days, certainly within ten. When this period is reached, mental excitement is succeeded by somnolence passing into stupor, contractions and rigidity yield to relaxation and paralysis, the pulse becomes slow, the respirations shallow and irregular, the coma deepens, all reflex movements are suspended, and death ensues. Excluding the prodromic period, the whole course of the disease may have been completed within seven to ten days. Death may also occur in these cases in the apoplectic coma, in the convulsions, or in the acute delirium which marks the onset of the inflammatory period. The cases do not all pursue the course just indicated. When the stage of depression is reached there may be a period of improvement, or the case may continue with the hemiplegia, the local paralysis, at a fixed point, the general condition, however, becoming much better. If the abscess is so situated in the hemispheres as not to involve the motor or sensory tracts, the symptoms of excitation will consist of delirium, epileptiform attacks, etc., and fever. The fever, as the author has witnessed, and verified the observation by *post-mortem* examination, may be intermittent, and, although somewhat irregularly so, be regarded as a genuine intermittent, and treated with quinia. The period of silence is rather a remission than a complete cessation of all morbid phenomena. As already indicated, some weakness or paralysis, lowered sensibility, defect of language, or impairment of mind remains. The abscess has been inclosed in its limiting membrane, and cut off from present mischief. In one case observed by the author, the patient so far improved in condition as to resume his occupation after a serious illness, but he still suffered from headache and vertigo and dimness of vision, and he experienced a remarkable change in his mental state: having been silent and reticent before, he became extremely talkative and communicative. This fact is all the more remarkable, since the abscess occupied the right anterior lobe. The period of silence is of variable duration, lasting from a few weeks to several months, during which the patient may be cut off by some intercurrent disease. There seems to be a relation between abscess of the right hemisphere and pneumonia. This period may be suddenly terminated by the abscess bursting into the ventricle, or at the surface of the hemisphere, which will be announced by violent convulsions, coma, and insensibility. Usually the end of this period is announced by an attack of intense headache, soon followed by drowsiness, and terminating in coma, or by convulsions and coma, or more slowly by a new meningitis. Not all cases of encephalitis pursue the defined course just described. The formation of the abscess may be quite latent, and no symptoms attract attention until convulsions and coma announce the end. Various forms are described by systematic writers, thus: the *meningeal form*, in which the fever is high, the delirium acute; the *comatose form*, in which the symptoms of excitation have been latent, and the early development of coma, dilated pupils, convulsions, and muscular resolution, indicate the extension of suppuration and early death; the *paralytic form*, in which limited abscesses occur in the motor ganglia at the base, and paralytic symptoms—hemiplegia, aphasia, and ocular disturbances—are present; the *apoplectic form*, in which sudden unconsciousness, followed by rigidity and paralysis, is the prominent feature; and the *epileptic form*, characterized by the predominance of eclampsia, succeeded by paralytic disorders.

Course, Duration, and Termination.—Notwithstanding the variability of the symptoms, encephalitis pursues a course not without uniformity. From the reception of the injury until the development of active symptoms is the prodromal period, of uncertain duration, from a few days to several weeks, even months. When the inflammatory process actually begins, the duration of the stage is about a week. Death may occur at this period. The period of silence is very variable also, and may be a few weeks' to several months' duration. A few hours or a day or two end this stage. The usual termination is in death. Recovery has taken place during the stage of inflammation, and by the discharge of pus spontaneously or by puncture.

Diagnosis.—The diagnosis involves the question of the seat of the abscess and the differentiation of abscess from tumor, from cerebral hemorrhage, and from meningitis. If the abscess is situated in the hæmispheres above the motor ganglia, there will be delirium and convulsions, and not contractions or paralysis; and, if in the region supplied by the left middle cerebral artery, amnesic aphasia will be present. If the abscess forms in the motor ganglia at the base, hemiplegia will be the prominent symptom; or paraplegia, should there be an abscess on both sides. If the abscess forms in the middle fossa of the skull, about the sella turcica, and involves the crus cerebri, there will be paralysis of the extremities on the opposite side, and of the third nerve on the same side. If the abscess occurs in the neighborhood of the pons, so as to impinge on one side, there will be a crossed paralysis of the facial on the same side and of the members on the opposite side. Abscess of the cerebellum gives rise to incoördination of muscular movements, vertigo, vomiting, amaurosis, and convulsions. In abscesses of the base, the cavernous sinus is compressed, and hence there will be present swelling of the eyelids, injection of the conjunctiva, and epistaxis. On ophthalmoscopic examination, the retinal veins are swollen, tortuous, and the disks are congested and stuffed (choked disks), but, in the further progress of the cases, white atrophy ultimately results. In abscess of the base and cerebellum, the retinal congestion occurs earlier and is more pronounced. There is no symptom of tumor which may not occur in abscess, but still a distinction may often be made. Tumor develops more slowly than abscess, and is unaccompanied by fever. The symptoms are continuous in cases of tumor, and there is no period of silence. Abscess is often connected with injury, with caries of the bones, disease of the ear and nose; tumor develops without any cause. Between the apoplectic form of abscess and cerebral hæmorrhage there is no well-marked distinction except as to termination, which resolves the doubts. The other forms of abscess do not come into relation to cerebral hæmorrhage. Abscess of the cortex and meningitis present the same symptoms of irritation followed by depression, but in the latter there is no period of silence followed by relapse.

Treatment.—The stage of inflammation requires active measures to prevent further mischief, as the remedies already advised for acute meningitis. Ergot, quinine, and chloride of barium (liq. barii chloridi ♏ xx every four hours) are the most efficient means of preventing the migration of the white corpuscles and the diapedesis of the red. When suppuration occurs, it is good practice to check the formation of pus, and the collateral œdema and hyperæmia, by full doses of quinine. The propriety of trephining, or of puncturing the brain, to favor the exit of pus, is a question of purely surgical interest, into the discussion of which we do not purpose to enter.

INTRA-CRANIAL TUMORS.

Definition.—The term *intra-cranial tumor* is a more correct designation than cerebral tumor, for it includes all neoplasms so situated as to affect the contents of the cranium. The term cerebral tumor takes into consideration, if restricted to its proper meaning only, tumors of the cerebrum, and not those of the meninges, of the vessels, etc. By the term *tumor* in this connection are intended all kinds of growths or outgrowths, and it is not confined to its merely technical signification.

Causes.—Intra-cranial tumors are usually divided into four groups: the vascular; the parasitic; the diathetic; and the accidental. Tumors are more common in men than in women, simply because men are more exposed to the influences producing them. Injuries excite osseous and connective-tissue hyperplasia, and a violent strain may be the cause of an aneurism. The diathetic tumors are in part transmitted by inheritance, in part acquired.

Pathological Anatomy.—Of 551 cases of *aneurism* in various parts of the body, only seven were intra-cranial.[*] The arteries of the base only are concerned, for a miliary aneurism is not a tumor in the sense in which that term is here used. The internal carotid and its branches are most frequently affected; in a total of 172 cases, 116 were of these vessels, and 53 were of the vertebro-basilar arteries. Taking individual arteries, we find that in a collection of 142 cases there were forty-one of aneurism of the middle cerebral, forty of the basilar, twenty-three of the internal carotid, fourteen of the anterior cerebral, eight of the posterior communicating, seven of the vertebral, four of the posterior cerebral, three of the inferior cerebellar, and two of the anterior communicating. As respects the side of the brain, the left is more frequently affected by aneurism. In a collection of sixty cases, thirty-five were on the left and twenty-five on the right side.[†] As regards size, intra-cranial aneurisms vary greatly, those of the anterior and middle cerebral artery attaining to the greatest size. From a pea to a pigeon's-egg is the usual size, but they may attain to the dimensions of a hen's-egg. The *parasitic tumors* consist of the cysticercus cellulosæ, or the echinococcus. The former are small vesicles the size of a pigeon's-egg, composed of a transparent wall and pellucid contents. They are found often in large numbers in the gray matter of the hemispheres, in the pia mater, and, as the author has seen, on the floor of the fourth ventricle. The echinococcus cyst is larger, often solitary, and never exceeding three to five. It has a tougher investing membrane, but transparent contents in which can be seen the scolex with its hooklets (Davaine). The *diathetic tumors* are cancer, syphilis, and tubercle. Cancer is a very frequent form of tumor, and, although at one time was supposed never to occur as a primary disease, is now known to be often primary. According to the statistics of Lebert, of forty-eight cases of cerebral cancer, thirty-five were primary. According to Bacon,[‡] only ten in seventy-three cases were primary. Ogle[§] finds that thirteen out of twenty-five occurred in the brain alone. When secondary, there are several nodules; when primary, a single one, which is usually quite separated from the tissue in which it is imbedded. The largest tumors are those growing in the hemispheres, an example of which the author saw, having the dimensions of the closed fist. The form is usually encephaloid, rarely scirrhous, still more rarely colloid and melanoid. The position of the cancer, named in the order of relative frequency, is the hemispheres, the cerebellum, corpus striatum, optic thalamus, and pons. Cancer of the orbit, of the scalp, or of the cranial bones, may grow inwardly to the brain ; on the other hand, cancer of the brain tends to develop outwardly. The form of *syphilitic tumor* is a gumma of the dura, and may occur at the convexity, but its favorite site is in the middle fossa of the skull, about the sella turcica. They do not attain to great dimensions, rarely exceeding a walnut, and more frequently having the size, as also the shape, of an almond. *Tubercle*-masses consist of an aggregation of cheesy nodules, and vary in size from a pea to a walnut. The most frequent situations are the cerebellum and the hemispheres, and much less often the corpus striatum and optic thalamus. The group of intra-cranial tumors called *accidental* contains glioma, sarcoma, steatoma, myxoma, psammomata and exostoses. Gliomata develop from the neuroglia, and are hard or soft, according to the quantity of granular and cellular contents and fibrillæ. They are very vascular, and hence may be accompanied by considerable hæmorrhage. They are found in the hemispheres, in the gray and white matter, and may be attached to the membranes. Of the sarcomata, there are several varieties ; they may adhere to the meninges, or develop in the hemispheres, or in the motor ganglia, at the base. Lastly, the cholesteatoma, which grows from the arachnoid or pia mater, and is found on the hemispheres and in the posterior fossa, attains by the aggregation of several smaller tumors sometimes to the size of a goose-egg. A growing tumor affects the parts in its immediate neighborhood by the irritation which its presence excites, and by destruction of tissue effected by pressure. Neuritis and ultimate softening and disintegration of nerves impinged on, inflammation, absorption, and softening of the adjacent portion of cerebral matter, are pathological results of the proximity of a tumor to the intra-cranial organs. Besides the local effect, a growing tumor increases the pressure of the organs, and causes a displacement of the movable contents of the cavity, the blood and cerebro-spinal fluid, and an approximation of the perivascular lymph-spaces. Pressure on the sinuses interferes with the venous circulation.

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[*] "Transactions of the Pathological Society," vol. vii, *op. cit.*

[†] The above statistics of intra-cranial aneurism were obtained from an article on "Aneurism of the Brain" by the author, published in the "American Journal of the Medical Sciences," October, 1872. The statistics of Lebert, of Durand, and of Gougenheim, were analyzed in this article.

[‡] "On Primary Cancer of the Brain," London, 1865, pamphlet.

[§] Reynolds's "System of Medicine," vol. ii.

Symptoms.—The symptoms produced by intra-cranial tumors are divisible into two classes : those common to tumors in all situations ; those caused only by tumors in particular situations. In the first group are headache, vertigo, amaurosis, convulsions, and mental disorders ; in the second, aphasia, strabismus, ocular paralyses, and hemiopia, tic-douloureux, facial spasm or paralysis, deafness, incoördination, vomiting, crossed paralyses, etc. Headache is of so persistent and violent character that Ladame* holds it has high diagnostic importance. It consists of paroxysms of acute pain and a constant feeling of uneasiness. The pain is increased by jarring the head, by tapping even gently, and by a full inspiration. Sometimes the position of the pain indicates the site of the neoplasm ; as pain in the forehead, when the tumor is in the anterior lobe ; in the occiput, when the tumor is in the cerebellum. Vertigo comes on usually some time after the headache, and is present to a greater or less extent in all cases, but is more pronounced in the case of tumor of the cerebellum. Slight fainting-fits, with or without the most transient loss of consciousness, and accompanied by intense vertiginous sensations, occur in many cases. Early in the development of the tumor the vertigo subsides on assuming the recumbent posture and closing the eyes, but later the vertigo comes on severely when the position is horizontal, the bed and all objects being in more or less rapid motion. In advanced cases, the vertigo is so severe as to prevent walking, or at least to render it difficult and uncertain. Amblyopia and amaurosis are also symptoms of tumor in any situation, for, as Hughlings Jackson well says, "so far as the production of optic neuritis by intra-cranial disease is concerned, the position of the disease seems to be of little consequence, and there is nothing very peculiar in its nature, except that it is usually coarse." Graefe held that the retinal changes were due to direct pressure on the cavernous sinus, the return of blood from the orbit being thus prevented, but Lancereaux and others demonstrated that the pressure was not sufficient to do this in the case of many tumors situated at a distance. Neuro-retinitis, then, is a general symptom of intra-cranial tumor, but the retinal and orbital changes may also have special significance. Convulsions, local and partial, may furnish topographical indications, but general convulsion may accompany tumor in any situation, unless we except the pons Varolii, on the dictum of Ladame. Greater or less departure from a healthy mental state is observed in all cases of tumor, and those involving the gray matter probably affect the mind more, but actual insanity has been observed in about one third only. In many cases, changes of disposition occur, usually in the way of moroseness, irritability, and depression ; in others, the faculties seem enfeebled, the power to apply the mind to any intellectual effort wanting: but the author has seen a case in which the patient, a clerk, developed a great capacity for the acquisition of languages during the time when the tumor, which occupied the posterior lobe of the left hemisphere, was forming. Eccentricities of conduct, delusions, and various other forms of mental derangement, accompany tumors of the brain, and a considerable proportion of such cases enter asylums for the insane. The symptoms which serve to indicate the position of the neoplasm are very important, and often extremely characteristic. The existence of amnesic aphasia—loss of the memory for words—strongly implies lesion of the left anterior lobe, fissure of Sylvius or island of Reil, or of the parts supplied by the left middle cerebral. A tumor of the cortex of either hemisphere may give rise to convulsive movements in the hand and arm of the opposite side, with or without general convulsions and loss of consciousness, and, if posterior, will involve sensibility as well as motility. A tumor impinging on the motor centers (corpus striatum, thalamus opticus, etc.) will produce first, irritation—spasmodic contraction and rigidity on the opposite side, and next depression by destruction of tissue—paralysis on the opposite side of the body. A tumor so situated as to impinge on the crus cerebri and the third nerve will produce symptoms differing according to the injury done ; if the result is irritation, irregular movements of the eye (nystagmus) on the same side, and rigidity and contraction in the muscles of the opposite side of the body ; if the result is destruction of tissue, there will be ptosis, convergent strabismus, and dilated pupil in the eye of the same side, and paralysis of the muscles on the opposite side of the body. If a tumor is so situated as to compress the optic nerve at the outer side of the chiasm, the field of vision will be narrowed to a degree corresponding to the extent of the injury, and destruction of the chiasm would cause blindness. Irritation of the olfactory would give rise to strange smells, and destruction of the nerve to loss of the function. Tumors at the base may involve several cranial nerves, causing disturbances of great significance, either of irritation or loss of function. If the fifth nerve is irritated, tic-douloureux will be the result ; but, if the nerve is destroyed, there will be anæsthesia of all the parts to which the nerve is distributed. A tumor of the pons can be diagnosticated by the implication of the fourth, fifth, and sixth nervés on the same side, and by disorders of motility and sensibility on the opposite side, and by the absence of convulsions (Ladame). A tumor of the medulla oblongata causes disturbances in the important functions whose centers are located here—in speech, deglutition, respiration—causes disorders of sensibility and motility on the opposite side and of the face on the same side ; causes vomiting, constipation, and paralysis of the bladder, etc. Tumors of the corpora quadrigemina affect the motions of the eyes, set up double optic neuritis, and cause paralysis on the opposite side of the body. Tumors of the cerebellum disorder the function of coördination, especially of those movements requiring the eyes to guide them, cause excessive vertigo, and difficulty in maintaining the upright position, optic neuritis and early extinction of vision, and general convulsions. Tumors at the base, by pressure on the cavernous sinus, interfere with the return of blood from the facial vein, and cause swelling of the eyelids, bleeding at the nose, and fullness about the orbit. A growing tumor, by displacing the cerebro-spinal fluid through the internal and external sheath of the optic nerve, renders the eye more prominent, and, by pressure on the cavernous sinus, maintains congestion of the orbital and retinal veins ; and hence, although retinitis occurs when tumors are in the hemisphere anywhere, it will develop earlier and more severely in the case of tumor at the base. It has been ascertained that considerable atrophy of the optic disks is not incompatible with fairly good vision. The general condition of the subjects of intra-cranial tumor may be very good. When there is vomiting, there will be wasting from an inability to retain the necessary aliment. If the tumor is cancer, the peculiar earthy hue, the wasting, and emaciation will soon be manifest.

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* "Symptomatologie und Diagnostik der Hirngeschwülste," Würzburg, 1865.

Course, Duration, and Termination.—Obviously, there can be no uniformity in the course of tumor. The symptoms are, at first, very indefinite, and, in the case of some of them, at least months are occupied in developing any well-defined ailment. A persistent headache, vertigo, alterations of demeanor, are first noticed, and gradually the character of the case becomes known. Tumors situated in parts of the brain that are well called "indifferent" may never cause characteristic symptoms, but usually now a correct diagnosis may be made if the case is thoroughly evolved. The duration of tumor varies from two to three months, up to five or more years. Unless the tumor is syphilitic, or possibly aneurismal, there can be but one termination. Some end in a convulsion, or rather in the secondary coma which follows it; others are cut off by an intercurrent disease, and notably pneumonia, or by cerebral hæmorrhage, or by acute meningitis. Aneurism terminates by rupture, unless by treatment its consolidation may be effected. Before the access of the final coma a remarkable degree of somnolence is observed in some cases, sleep continuing for several days at a time uninterruptedly.

Diagnosis.—The determination of the position of the tumor has been sufficiently considered. Can a diagnosis be made of its nature? Aneurism occurs in adults or the old; in those who continue to have good health, and who are not affected by a diathesis or an hereditary ailment. Vomiting is not usual; the cranial nerves are early paralyzed, and on the same side as the tumor; the mental functions are not often affected; epileptiform seizures do not occur, and the termination is by an apoplectic attack. An aneurism of the internal carotid within the carotid canal will cause protrusion of the eye by obstruction of the cavernous sinus, and may be accompanied by an audible *bruit*. A tubercular tumor is usually accompanied by the evidences of tubercular deposit elsewhere. The subject is young, and evidences of hereditary taint may be present; it is situated deeply, often in the indifferent districts, and does not produce disturbances in the cranial nerves. Syphilitic gummata have a tendency to form in the middle fossa, and to affect the *crus cerebri* and third nerve, and are usually coincident with external lesions. Echinococci or cysticerci are accompanied by numerous epileptic attacks, at first without any injury, but subsequently the mind becomes torpid, and passes into dementia. Local paralysis and hemiplegia are uncommon. The distinctions between tumor and abscess have been given in the article on abscess. The differentiation between obstruction of the cerebral vessels and tumor may often be a matter of extreme difficulty. Tumor may appear at any age; thrombosis is usually a disease of advanced life. Thrombosis is accompanied by and due to chronic arteritis; tumor is not related to general arterial changes. Tumor is characterized by intense headache; thrombosis by less severe and persistent. Tumor is generally accompanied by epileptiform attacks; thrombosis by apoplectic. Tumor affects the cranial nerves, and causes localized paralysis; thrombosis never produces such results.

Treatment.—There are two remedies which ought always to be used —iodide of potassium and ergot; for, although only syphilitic and possibly aneurismal tumors are remediable, the case under treatment may be one of them. Scruple-doses of the iodide of potassium should be given until iodism is induced. If no improvement is then manifest, it need not be continued. A drachm or two of the fluid extract of ergot four times a day may properly be given for several weeks succeeding the iodide. The repetition of these remedies will depend on the results of their first administration. They may effect a cure of the syphilitic and vascular neoplasms.

APHASIA.

Definition.—Inability to use spoken language or to give vocal utterance to ideas is designated *aphasia*. The defect may consist in a loss of memory of the words by which ideas are expressed, when it is called *amnesic aphasia;* it may consist, not in forgetfulness of the words, but in an inability to combine the different parts of the vocal apparatus for vocal expression—*ataxic aphasia*. When the defect involves written language, and consists in an inability to recognize and make the signs by which ideas are communicated in written language, it is named *agraphia*, and this may be either *amnesic* or *ataxic*—the former being a mental defect, the latter an affection of the muscular apparatus, known as writer's cramp. Amnesic aphasia exists to a variable extent, and may, indeed, involve but a limited number of words. *Paraphasia* is a term proposed by Kussmaul[*] to signify the mental state in which the wrong words are used, or unintelligible expressions employed to express the idea. There may also be a *paragraphia*—a state in which wrong or meaningless written signs may be used to express the idea.

Pathogeny.—Aphasia and its various modifications are associated with a number of intra-cranial lesions; with occlusion, either by thrombosis or embolism of the vessels; with cerebral hæmorrhage; with encephalitis and abscess; with meningitis; with the various forms and varieties of tumors; and it may be a merely mental and moral condition. Associated with so many and varied maladies, and occasionally existing alone, as the sole evidence of disease it is necessary to give the subject independent and separate consideration. We can not occupy space with an extended historical account of the progress in the knowledge of this peculiar condition, but we may state some facts, and begin by saying that to Gall unquestionably belongs the credit of first suggesting the position of the language faculty. He says, "I regard as the organ of verbal memory that cerebral part which rests on the posterior half of the roof of the orbit."* Thomas Hood, quoted by Hammond,† so long ago as 1822 described accurately a case of aphasia. Bouillaud published a work in 1825 to prove the correctness of Gall's doctrines that the language faculty was situated in the anterior lobes. Marc Dax in 1836 made the remarkable statement that, in cases of aphasia, the paralysis was on the right side and the lesion on the left, thus limiting the seat of the language faculty to the left frontal lobe. The next and most important step was that taken by Broca in 1861, who sought to prove by cases that "the integrity of the third left frontal convolution, and perhaps also the second, is essential for the development of the power of articulate speech." The observations on man seem to be confirmed by the experiments of Ferrier ‡ and Fritsch and Hitzig, which show that electric irritation of a corresponding part in animals is followed by "alternate opening and closure of the mouth, with movements of the tongue." It seems to be now pretty definitely settled that lesions of the region supplied by the left middle cerebral artery, notably the island of Reil, the third convolution, and the neighboring part of the corpus striatum, are those accompanied by the various forms of derangement included under the term aphasia. Hence it is that right hemiplegia and aphasia are so often associated. First in point of importance are lesions of Broca's convolution, next those of the island. Why the left hemisphere should be alone the seat of such a faculty, and not the right, has received various explanations, but that offered by Broca is probably the most nearly true—that the left hemisphere is earlier and more rapidly developed, receives more blood, and is therefore first and chiefly instructed, whence the greater skill and education of the right hand. Cases of left-handed persons becoming aphasic from disease of the right hemisphere have been reported. There are cases of aphasia in which the power to write correctly is retained—aphasia without agraphia. In other cases there is an absolute inability to communicate ideas by written signs, all attempts resulting in a meaningless scrawl. The two functions must therefore possess different centers and yet be in close proximity. Sign-speech, or the power to express ideas by signs, or sign-language, may or may not be simultaneously affected with the language faculty. As patients may or may not be conscious of the defect, there are consequently an *amnesic amimia* and an *ataxic amimia*. As amnesic aphasia may coexist with retention of the power of written language, by which the intellect may be tested, it has been demonstrated that the existence of aphasia is not incompatible with the full possession of the intellect in all other respects. A number of cases have now been reported in which amnesic aphasia was the sole lesion. The importance of this observation, from the medico-legal point of view, is very great. On the other hand, it is generally true that the mind is weakened or impaired in other respects, so that the presence of aphasia is *prima facie* evidence of mental impairment. Aphasics are often very curiously damaged. A musician could not read the musical notes, but could play by ear; on the other hand, Lasègue saw a musician with both aphasia and agraphia, who could write down notes that he heard (Kussmaul); others can not count money, or distinguish the uses of table-utensils.

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[*] Ziemssen's "Cyclopædia," *op. cit.*

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* Gall's "Works," vol. v, p. 11, translated by Winslow Lewis, M. D.

† "Diseases of the Nervous System," *op. cit.*, p. 178, sixth edition.

‡ "Functions of the Brain," American edition, 1876, p. 143.

Course, Duration, and Termination.—The forms of aphasia pursue a course parallel to the malady with which they are associated, as a rule, but sometimes aphasia ceases before the disease, or continues after the disease has disappeared. Aphasia may be hysterical or due to curable disease, as syphilis, or it may be produced by reflex disturbance of function, as parasites in the intestines, or constipation. The duration will be brief under these circumstances, and the termination be in recovery, if right means are used. As regards the influence of permanent lesions, the results depend somewhat on age, for in children extensive injuries to the language center may be overcome by training, but in the aged limited lesions are fixed in their effects. Simple amnesic aphasia is more favorable, and ataxic aphasia is less favorable, as regards the prospect of recovery. The longer the condition of aphasia has existed, the less the prospect of recovery. The case is still less favorable when the aphasic state is increasing *pari passu* with the disease on which it depends.

Treatment.—The local disease must be removed if of a curable kind. If the case is one in which aphasia persists after the disease on which it depended has been removed, much may be done by suitable training. An admirable example of the results which can be obtained by rightly directed effort is that of Bristowe,* of a Canadian in St. Thomas's Hospital, perfectly aphasic, whose speech was entirely restored in eight months by a course of carefully conducted speech-lessons given by Dr. Bristowe. These Lumleian lectures deserve the attentive study of those who desire to have a truly scientific and philosophical knowledge of the subject.

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* The Lumleian Lectures, on the "Pathological Relations of Voice and Speech," London "Lancet," June 21, 1879.

VERTIGO.

Definition.—*Vertigo* is not properly a substantive disease, but it may be the only symptom of the morbid state to which it is referable. It is a subjective state, in which the individual affected, or the objects about him, seem to be in rapid motion, of a rotary, circular, or to-and-fro kind. In common language vertigo is known as dizziness.

Pathogeny and Symptoms.—Vertigo may arise from centric or eccentric causes; hence it may be cerebral, auditory, cardiac, or stomachal. One form of epilepsia mitior is vertiginous, or the manifestation of the seizure consists in an attack, more or less violent, of vertigo. Other affections of the brain have the symptom vertigo at various times in their course. These have been, or will be, alluded to at the proper time, and hence do not require further statement here.

AUDITORY VERTIGO is named after its discoverer—*Ménière's Disease.* It has long been known that puncture of the auditory nerve in rabbits is followed by rotary movements of the animal. An inflammation of the middle ear will often excite convulsions in children. Injecting water in the ear will induce vertigo. Ménière described, in 1861, a sudden, excessive vertigo, produced by an hæmorrhagic extravasation or some form of exudation into the semicircular canals of the internal ear. This is the true Ménière's disease, but vertigo is associated with other aural lesions. Thus, it may occur in the course of an otorrhœa, accompanied by noises, beating, singing, and other troubles of the ear. Again, without any known disease of the ear, the patient may be disturbed by temporary attacks of vertigo, occurring from time to time, until at last it becomes a permanent condition. In still other cases an individual in full health, apparently, is suddenly seized with a formidable attack of apoplectiform vertigo. Many of the cases of temporary or habitual vertigo, and of apoplectiform attacks which appear to be independent, in reality coexist with disorders of the ear which pass unnoticed. At the outset Ménière's vertigo manifests itself in short, sudden attacks, separated, by long intervals free from disturbance. If the malady is aggravated, the attacks approximate, and finally become constant but diversified by paroxysmal exacerbations. In the worst cases the victims are compelled to remain in a horizontal position, which moderates the distress. But even then they are in an unstable condition, and must preserve a most quiet attitude, with closed eyes, if they would avoid seizures. The forms taken by the vertiginous sensations are various; sometimes it is a see-saw, a gyratory motion, right or left; sometimes a vertical whirl, a sensation of rising and falling, like the swell of the ocean, etc. It is not surprising that persons so affected, for months or years, experience changes in character—become morose, irritable, suspicious.

In other cases the vertigo, although habitual, is less severe, and those affected learn that severe seizures may be avoided by keeping a position of fixed attention, and rigidity of the head and members, somewhat inclined against the direction to which the vertigo carries them.

The permanent vertiginous sensations are often accompanied by various subjective noises in the ears. They assume every kind of tone—whistling, singing, beating, roaring, noise of escaping steam, etc.

Besides the subjective sensation of falling, in certain cases a sudden access of vertigo destroys the voluntary control, and the individual is precipitated to the floor. In a portion of these the sensation experienced is that of rapid rotation, but a fall may be prevented if some support can be seized in time. In others, an irresistible propulsion forward precipitates them on one side or the other, or they fall as if struck a violent blow on the nucha, and sometimes with sufficient force to be injured in coming down. The fall is forward, rarely backward, and obliquely to one or the other side, sometimes to the side of the affected ear, sometimes to the other side. This lateral propulsion, Gowers holds, is diagnostic of Ménière's vertigo. At the moment of the attack, the patient, although deprived of voluntary control, does not lose consciousness. Nausea and vomiting occur in many cases at the end of the seizure; in others, only nausea is experienced. Some glairy mucus and bilious matters are brought up by vomiting, and but little food, as a rule.

Not all cases of Ménière's disease become habitual and constant. In many cases the attacks are isolated, and separated by long intervals of entire freedom from the sensations. The attacks may present the most violent and characteristic features, or may be mild. The vertigo, the gyratory movements, and the vomiting, may take a predominant position, but they do not constitute a special form of the disease. Two distinct forms may be recognized: one, consisting of a constant state of vertigo, diversified by paroxysms; the other, made up of isolated seizures, separated by periods of good health.

There is no constant anatomical lesion associated with auditory vertigo. In many simple cases a sero-sanguineous exudation has been found in the semicircular canals; in other cases there have existed complicated lesions, resulting from traumatism. Various lesions of the internal ear have been associated with labyrinthine vertigo, such as catarrh of the drum and of the mastoid cells. Probably any trouble of the ear, external or internal, may produce the phenomena of Ménière's disease, by modifying the pressure on the terminals of the auditory nerve. These phenomena may also be caused by a neuritis or perineuritis of the auditory nerve, and have been associated with facial paralysis, or other disorders of the facial or chorda tympani. When bulbar lesions produce vertigo, there are neuralgic pains in the distribution of the fifth nerve, and the same anatomical relationship explains the nausea and vomiting, the nucleus of the fifth nerve and of the pneumogastric having close connections.

STOMACHAL VERTIGO is much more frequent and more important, therefore, than Ménière's disease. There can be little doubt that it is frequently mistaken for cerebral disease. It is a misleading error to suppose that stomachal vertigo is always associated with pronounced stomachal disorder. There are cases in which the disturbance of the intracranial circulation—the vertiginous sensations—constitute the only apparent evidence of stomachal disturbance. In such cases the symptoms always come on during the stomach or intestinal digestion. In other instances there are felt, after eating, a sense of fullness and repletion of the stomach, and hebetude of mind. Again, the attacks of vertigo are experienced by some persons after eating an article of food especially indigestible, and are infrequent. Usually the cerebral symptoms occur in the course of well-marked and long-standing stomach and intestinal disease. Always after eating heartily there are heaviness and oppression, acidity and pyrosis, and frequent eructations occur; the abdomen some hours after meals is distended with flatus, and constipation is the rule. These subjects are usually of full habit, and tend to obesity, have acid urine loaded with urates, and are rather sluggish, physically, although active in their mental habits. Eating habitually more than is needed, and leading sedentary lives, unoxidized products accumulate in the blood, and uric acid and urates are largely present in the urine. Much flatus in the intestine is coincident with the vertiginous attacks in some subjects.

Stomachal vertigo may occur under opposite states of the intracranial circulation; under anæmia or hyperæmia, the latter more frequently. The evidences of hyperæmia are a rather flushed face, injection of the conjunctiva, rather full retinal vessels, and occasional epistaxis. The anæmic subjects are pale, the sclerotics pearly white, and the retinal vessels small. The mechanism of the vertigo is complex. There are two factors: one consists in the toxic effect of the imperfectly oxidized materials which accumulate in the blood; the other is reflex. An impression made on the end organs of the pneumogastric in the stomach is reflected over the sympathetic ganglia or over the auditory, including its terminals in the semicircular canals. The attacks of vertigo occur in two forms: acute and severe, chronic and light.

The acute and severe attacks occur during the digestion of a full meal, or of some especially indigestible material which lingers in the stomach. Suddenly there is felt an odd sensation in the scalp, a creeping coldness, a sense of constriction, surrounding objects appear to go round and round with all the varied movements of the kaleidoscope, the individual reels and seizes some object or person for support, he has a seasick feeling, more or less intense, and may vomit; his face is pale, pulse feeble, and his vision is blurred. If vomiting occur and the stomach is entirely emptied, the symptoms subside. If there is nausea, merely, the sick feeling will presently subside and with it the vertigo, to return, it may be, in a few minutes, and be repeated several times, until the digestion is completed. In other cases the nausea persists until some intestinal uneasiness, followed by a copious evacuation, occurs. The consciousness is not usually lost in such attacks, but the mind is dazed; in some cases there are hallucinations and illusions during the period of vertigo. The depressing effect on the vaso-motor system is exhibited, not only in the feeble circulation and the sudden pallor, but the surface is bedewed with a cold sweat, or sweating is limited to one side of the head or of the body, or to one extremity. These acute attacks are doubtless entirely reflex.

The chronic form of stomachal vertigo is manifested by nearly constant headache, noises in the ears, and vertigo, which is daily in occurrence, or nearly so. In the most typical cases, a feeling of uncertainty and unsteadiness comes on after breakfast and increases until actual vertigo develops, reaching its maximum some time after dinner. At no time is there a degree of unsteadiness requiring support, but a feeling of reeling and dizziness, making an effort necessary to preserve the vertical position. Not all cases are nearly constant or daily. There may be intervals of entire exemption, caused by greater care in the selection of food, and by more perfect digestion. Although the vertigo may be only occasional, these subjects suffer from headache, uneasiness after food, acidity, and flatulence nearly constantly. There are a few cases in which, with severe vertigo, no trouble with the stomach appears to exist, but on close examination it will be found that digestion labors, and that there is a sense of oppression at the epigastrium.

The form assumed by the vertigo varies. It may be a sudden feeling of emptiness of the head, an apparent sudden deprivation of thought, volition, and consciousness. Sometimes there is a mist before the eyes, surrounding objects are blurred, the mind is confused, and the body reels. In other cases, the individual attacked feels firm, but all surrounding objects are whirling around him and around each other in inextricable gyrations. By closing the eyes and keeping perfectly quiet, the equilibrium may be maintained, but in some cases this stratagem only induces the sensation of rising and falling, of floating away, etc. When the attacks occur in bed or on the lounge, these objects whirl around, or float away, or rise and fall as on the ocean-swell. Various hallucinations of sight and illusions occur, but consciousness is not lost, and delusions are not produced.

While the attacks of vertigo, occurring during the course of the chronic form of the malady, are largely reflex, there is another factor of considerable pathogenetic importance. Imperfect digestion, especially the intestinal, lessened oxidation because of an excess of material to be acted on, or diminished supply of oxygen, and sluggish elimination, are concerned in the production of a morbid state of the blood, of special importance in this connection. The urine is acid in reaction, highly pigmented, and heavily loaded with urates and uric acid. The most highly specialized tissue will be the first to be acted on by such materials in the blood; hence the injury done to the brain and manifested in the headache, torpor of mind, etc., which are such prominent features in these cases of stomachal vertigo.

Cardiac Vertigo is a condition of anæmia of the brain, and is closely allied to fainting. The subjects of this malady are rarely free from vertiginous sensations on assuming the erect posture, and may be violently attacked on making any considerable exertion. They have a swimming sensation in the head, darkness falls on the eyes, and they become chilly and weak. Cardiac vertigo is associated with fatty heart and dilatation of the right cavities, conditions already described.

Cerebral Vertigo has received attention in connection with the several maladies of which vertigo is a symptom. It occurs as a symptom in cerebral anæmia and cerebral congestion; in cerebro-spinal meningitis, in which it is very pronounced in the stage of excitation; in tumor of the brain; in abscess of the brain; in cerebral hæmorrhage, as a prodrome; in sclerosis of the brain; in chronic endarteritis, with thrombosis especially; in epilepsy, chorea, and hypochondriasis; in chronic alcoholism, and chronic plumbic, paludal, and other forms of poisoning, etc.

Course, Duration, and Termination.—The conditions producing vertigo are so various that no uniform course can be laid down. Ménière's vertigo is usually incurable, as dependent on lesions that are not remediable. In some cases there is a gradual development of continuous vertigo from lesions of the ear, and so severe does it become that the victim is unable to pursue any occupation—to sit up, even. In other cases the attacks are paroxysmal, and occur in the course of chronic affections of the ear. The genuine Ménière's disease is a malady characterized by a sudden attack of vertigo, with nausea and vomiting so severe that the patient falls as if from an apoplectic attack. There has been no previous disease, and the change in the ear consists in an hæmorrhagic or sero-sanguineous extravasation into the semicircular canals. Such cases without any other lesions have, in a few instances, terminated fatally. Permanent deafness results, and afterward paroxysmal attacks of vertigo occur, or the vertigo may become a permanent condition. The fortunes of vertigo, associated with aural lesions, are those of the lesions: if the lesions are curable, vertigo ceases, but as a rule these cases are not hopeful.

Treatment.—The treatment of auditory vertigo resolves itself into the management of the aural lesions. Charcot[*] has recently called attention to the great value of quinine in full doses (ten to fifteen grains) in the treatment of Ménière's disease of that form characterized by the hæmorrhagic or sero-sanguineous exudation into the semicircular canals. Confirmatory experience has been reported by Ménière,[†] and by Féré and Demars.[‡] In 1875, say Féré and Demars, Charcot made known in one of his lectures at La Salpêtrière, the results of a new treatment which he had been using some months. He presented to the class in attendance a patient, who had many years been confined to bed with a vertigo nearly permanent, diversified by extremely severe paroxysms. She had subjective noises in the ear, which, joined to a purulent discharge from the external auditory foramen, left no room for doubt as to the origin of the disease. Charcot conceived the idea of modifying the ear-noises by the administration of large doses of quinine, which he gave during two months in doses of eight to fifteen grains a day. The roaring in the ears was, after a time, replaced by the noise produced by quinine; the vertigo diminished notably, and in two and a half months the patient was able to walk with the aid of a cane. Féré and Demars find that, when from eight to fourteen grains of quinine are given daily to the patients with auditory vertigo, the symptoms of the disease are notably increased. If, after eight to ten days' administration, the quinine is suspended, the *bruit* and the vertigo are then found to be much less than before the use of the remedy was begun. If the treatment is resumed again, a new exaggeration of the symptoms occurs, but usually much less than on the first trial, and, when again suspended, the improvement is found to be still greater. Excellent results have thus been produced. The author, from experience in one case only, is able to confirm the report as to the exceptional value of quinine in auditory vertigo.

In the treatment of stomachal vertigo, careful regulation of the diet is necessary. It is often extremely serviceable to begin the treatment by an exclusive milk-diet, and then to reconstruct the dietary, according to the condition of the individual subject. Arsenic—one or two drops of Fowler's solution, three times a day—is the best remedy. When the appetite is poor and the general state feeble, tincture of nux vomica is an efficient remedy. When there is much nausea, carbolic acid, with or without bismuth, is very serviceable. When there is gastro-duodenal catarrh, phosphate of soda is useful in a high degree. If the urine is loaded with uric acid and the urates, nitric acid or the potash alkalies, as the case may be, will prove to be very beneficial. Each case must be studied by and of itself. Besides the merely medicinal and dietetic treatment, much good is accomplished by regulated active exercise in the open air.

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[*] "Progrès Médical," 1875, p. 733.

[†] "Quelques considérations sur la maladie de Ménière," etc.

[‡] "Revue de Médecine," October 10, 1881, p. 796, *et seq.*

DISEASES OF THE MEDULLA OBLONGATA.

HÆMORRHAGE.

Pathogeny.—It is a rare event to have hæmorrhage occur in the medulla or pons, but cases have been reported. The conditions causing the hæmorrhage are doubtless very much the same as those of the brain, miliary aneurisms and atheroma being the chief factors. The larger aneurisms of the basilar artery may by rupture cause a hæmorrhage affecting this as well as other organs. The medulla is compressed by hæmorrhages from above, breaking through on to the floor of the fourth ventricle. These conditions are not now under consideration, the inquiry being restricted to hæmorrhage into the pons or medulla. The vessel affected in any case is small, the resulting clot is small, but there are usually several clots at the same time. They vary in size from a pea to an olive, but those examples of hæmorrhage in which the pons is simultaneously affected, or which occur in the pons, are much larger. One case is reported in which the hæmorrhage filled the whole of the pons, burst through on the left side, and also filled the fourth ventricle.* Another, in which the pons and fourth ventricle were invaded, and into the right crus cerebri there was also an extravasation.†

Symptoms.—If the hæmorrhage is large, vomiting usually occurs, consciousness is lost, there is complete muscular resolution, abolition of all reflex acts takes place, the breathing is sighing and irregular, becoming rapidly shallower, or is stertorous and noisy, the pupils are apt to be irregular, one large and the other minutely contracted, or both minutely contracted, death occurring in an hour or two, or in a day or two, in a deeply comatose state. There is a fulminant form, in which, hæmorrhage taking place in the medulla at or about the spasm-center, the patient falls with a cry into general convulsions, becomes comatose, and dies in a few minutes, or in an hour or two. Not all pursue this rapidly fatal course. A small clot may form on one side of the medulla or pons, there occur the usual symptoms of apoplexy, and the patient emerges from the condition of unconsciousness, after some hours or days, paralyzed as to motion and sensation on the opposite side (hemiplegia), or all of the extremities may be paralyzed more or less fully; or there may be a paraplegia, the arms escaping, but usually both upper and lower extremities are affected both as to motility and sensibility. There are usually paralyses of the cranial nerves—the third, fourth, fifth, the sixth, the seventh, etc.—and there may be paralysis of the body, on the opposite side of a unilateral lesion, while the cranial nerves are paralyzed on the same side. The breathing, owing to the proximity of the respiratory center, is irregular in rhythm, sighing, dyspnœic—often of the Cheyne-Stokes type. The action of the heart is not so much disturbed, but the pulse may be exceedingly rapid and irregular. Epileptiform convulsions are very usual and important from the diagnostic point of view, since Nothnagel's "spasm-center" is located in this organ, and hence clonic spasm would *a priori* be expected. Difficulty in swallowing (dysphagia) from paralysis of the palatal and pharyngeal muscles, and difficulty of speech from paralysis of the tongue (ataxic aphasia), and sometimes an obstinate singultus, are present in those cases emerging from the first coma. Albumen or sugar may be present in the urine.

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* Dr. T. S. Dowse, "Transactions of the Pathological Society," vol. xxvii, p. 7.

† Dr. J. W. Ogle, ibid., vol. xv, p. 9.

Course, Duration, and Termination.—As the facts above given sufficiently indicate, the course of hæmorrhage into the pons or medulla is rapid. Death may occur in a few minutes, in a few hours, or after several days. Very few recover in the damaged way above described. If such partial recovery ensue, the usual changes of an atrophic kind take place in the motor tract below the site of the hæmorrhage. The paralyzed muscles, innervated by the cranial nerves, it is probable, lose their electro-contractility in a few days.

Diagnosis.—It is often extremely difficult to distinguish between the coma and insensibility of hæmorrhage into the pons and the narcosis induced by opium or alcohol. There is no symptom produced by one which may not also accompany the other, but the antecedent history, taken with the group of symptoms as a whole, ought to conduct to right conclusions. The deviation of the head and eyes to the side of the intra-cranial disease, and from the paralyzed side, is a symptom of cerebral hæmorrhage, and not of opium or alcohol poisoning. Convulsions are uncommon in opium and alcohol poisoning, very common in hæmorrhage of the medulla. The pupils are often contracted in hæmorrhage, but never so minutely as in opium-poisoning. During the period of unconsciousness it may not be possible to diagnosticate between cerebral hæmorrhage and hæmorrhage of the pons and medulla, but the more frequent occurrence of convulsions, the vomiting, and the irregularity of respiration, may afford indications. Afterward the character of the paralysis, the manner in which the cranial nerves are affected, the paralysis of the palate, and difficulty of deglutition, the singultus, and the urinary derangements, serve for a ready and definite decision.

Treatment.—The management of hæmorrhage into the medulla or pons is the same as for cerebral hæmorrhage, which has been fully discussed.

OCCLUSION OF THE VESSELS OF THE MEDULLA AND PONS VAROLII.

Pathogeny and Symptoms.—The vertebrals and the basilar are the arteries affected. The mode of occlusion is by thrombosis and embolism, and the pathological results are such as have been described. The immediate effect of occlusion of the vertebrals is a sudden and intense anæmia, with or without loss of consciousness. There are paralysis of the tongue, palate, pharyngeal and laryngeal muscles, and paresis of the facial. Sometimes the ocular muscles, innervated by the third, and the masseters are also paralyzed, and usually there are great irregularities in the respiratory and cardiac movements. Paralysis of the four extremities, more frequently hemiplegia, as the left vertebral is the one ordinarily closed, results, and there may be, although not the rule, lessened sensation in the same parts. Death may ensue at once; the affected area, receiving no blood, ceases to functionate. In other cases, the first shock of the accident passes off, the paretic extremities contract and become rigid, and may remain in this state for many years. The symptoms produced by obstruction of the basilar are bilateral, and, as the glosso-pharyngeal and par vagum are paralyzed, there occur at the same time severe laryngeal and respiratory symptoms, with intense dyspnœa, and rapid carbonic-acid poisoning, and, if the immediate effects are survived, paralysis of the four extremities. The treatment of this malady is the same as for the same condition affecting the cerebral vessels.

ACUTE INFLAMMATION OF THE MEDULLA—ACUTE BULBAR PARALYSIS.

Pathogeny.—The changes resulting from inflammation of the medulla oblongata are the same as those of encephalitis: hyperæmia; exudation of serum, with its albumen and fibrin; migration of white corpuscles and diapedesis of the red; disassociation of the nerve-elements; changes in the neuroglia (multiplication of its cells)—the ultimate result being a spot of softening.

Symptoms.—The inflammation makes rapid progress. The onset of symptoms is sudden: a violent headache; intense vertigo; nausea and vomiting; excessive hiccough; inability or great difficulty in swallowing; toneless voice, or speaking difficult—and these symptoms appear without apoplectic symptoms or convulsions. As the medulla contains so many important centers within a narrow area, it is obvious that there may be much variety in the symptoms. If the pneumogastric nucleus is involved there will be embarrassed breathing, cyanosis, carbonic-acid poisoning, and the heart's action will be irregular, rapid, and weak. Paralysis usually invades the extremities, and varies much in extent: there may be hemiplegia, or all four extremities may be weak; sensation is not much affected. Neither tonic contractions of the muscles nor convulsions have been observed. The progress of the case is rapid. The difficulty of swallowing increases to absolute inability; the respiration is exceedingly irregular, and carbonic acid accumulates so that coma results, death occurring by failure of respiration.

Diagnosis.—It is probable that many cases diagnosticated hydrophobia were really examples of this disease. The distinction between inflammation, thrombosis, and embolism of the medulla, can not at present be made with certainty. While they all agree in symptoms of derangement of the important centers and nerves belonging to the medulla, myelitis of this part is not accompanied by apoplectic symptoms or convulsions, which belong to occlusion of the vessels.

Treatment.—The treatment is the same as that suggested for encephalitis.

CHRONIC INFLAMMATION OF THE MEDULLA—CHRONIC PROGRESSIVE BULBAR PARALYSIS.

Definition.—This disease is probably better known by the designation given it by Trousseau[*]—*glosso-labio-laryngeal paralysis.* This term was intended to express the main points in its symptomatology. Other names proposed are: *progressive muscular paralysis of the tongue, soft palate, and lips* (Duchenne[†]), and *progressive atrophic bulbar paralysis* (Leyden[‡]). *Chronic progressive bulbar paralysis,* the term proposed by Wachsmuth, and adopted by Erb, well expresses the seat and nature of the disease.

Causes.—The origin of the disease is very obscure. It occurs much more frequently in men than in women, and is a disease of advanced life, rarely occurring before forty. It has been referred to cold, to shocks, a blow on the neck, to rheumatism, to tertiary syphilis, to deep chagrin (Duchenne). It often coexists with progressive muscular atrophy (Friedreich[§]).

Pathological Anatomy.—Macroscopic examination may furnish only negative results. There may be changes of color and a dullness of appearance on section, and the medulla as a whole may appear to be shrunken,[‖] or harder or softer than natural, in places, but definite results are obtained only by microscopic examination. While the lesions in the medulla are so obscure to the naked eye, the nerves coming from this organ are changed in the most obvious way, especially the hypoglossal and facial. The important alteration, in regard to which observers are generally agreed, is an atrophy and degeneration of the multipolar ganglion-cells of the anterior cornua. The vessels are dilated, leaving vacuoles, there are numerous corpora amylacea, the cells (nuclei of hypoglossus, etc.) are crowded with pigment, the neuroglia overgrown (hyperplasia). Subsequently the cells disintegrate and disappear, whence the marked decrease in size. The nerve-roots and the nerve-trunks are also much changed, the nerve-fibers having undergone fatty degeneration, the neurilemma sclerosed, and the axis cylinder wasted till it is barely visible, and only a mass of connective tissue left. The most advanced changes are found in the hypoglossal nucleus ; next, the spinal accessory and the par vagum are attacked, and the facial and glossopharyngeal are more or less damaged, and, according to Clarke, the nucleus of the fifth is invaded to some extent. Similar lesions occur in the brain and spinal cord—throughout the whole extent of the cord, in a case described by Lockhart Clarke, which, however, was accompanied by progressive muscular atrophy.

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[*] "Clinique Médicale," vol. ii, p. 274.

[†] "D'Électrisation localisée," second edition, p. 641.

[‡] Quoted by Erb, Ziemssen's "Cyclopædia," vol. xiii.

[§] "Ueber, progressive Muskelatrophie," Berlin, 1873, cap. ix, s. 322.

[‖] Lockhart Clarke, "Medico-Chirurgical Transactions," vol. lvi, p. 103.

Symptoms.—The approach of the disease is very insidious. Headache felt in the occiput, some giddiness, a feeling of choking in attempting to swallow, a sudden inability to speak (Cheadle), are the symptoms first observed. The voice is not lost, but it has a nasal tone from the paralysis of the palate, and there is great indistinctness in speech because of the loss of power in the tongue and lips, the labial consonants not being pronounced. The tongue can not be protruded, and it wastes, becoming soon distinctly smaller. The food collects about the teeth and the cheek, so that the fingers are needed to dislodge it. The saliva dribbles from the mouth, the lips hanging limp and immovable. The taste is much less distinct or entirely wanting. It is a matter of great difficulty for the patient to get the alimentary bolus back into the pharynx. The efforts at swallowing excite coughing and suffocative attacks, and liquids are forced back through the nose. The palate and pharynx are so little sensitive that no reflex movements are caused by irritating them. The soft palate hangs limp and motionless in the fauces. When the disease reaches this point the appearance of the patient is eminently characteristic : the paralyzed lips and muscles of the face below the eye, their fibrillary trembling, and their motionless state in laughing, the flow of the saliva, the fatuous expression, the nasal speech, the inability to sound the labials, the choking in swallowing, the return of liquids through the nose, form a striking picture which no one can fail to comprehend. It is the sad fate of these patients to preserve their mental faculties, except that they become somewhat more emotional than formerly, and to continue conscious of their condition. The disease is truly *progressive*—the symptoms already described grow worse in every way—speech becomes less and less intelligible, swallowing more and more embarrassing and difficult, and the saliva increases in viscidity and quantity, the patient requiring a handkerchief constantly to absorb it. Other and more formidable symptoms now come on. The extension of the disease to the pneumogastric nucleus causes a paralysis of the muscles of the larynx, the voice is lost after preliminary weakness and huskiness, the respiratory muscles get weak and the lungs can not be expanded, and presently there are experienced oppression, heaviness of the chest, and constant dyspnœa, with paroxysms of a suffocative character, excited by the presence of mucus in the throat, by attempts of sneezing, coughing, or swallowing, or by the lodgment of some particle of food in the larynx. At the same time the action of the heart becomes irregular and weak, and attacks of præcordial oppression with a sense of impending dissolution occur. The condition of the patient is now truly pitiable. The mind is clear. The impossibility of swallowing leads to a rapid failure of strength, and, the digestive organs remaining unimpaired, an intolerable sense of hunger is felt. The termination may now be in a sudden failure of the heart, in an attack of pneumonia from lodgment of a foreign body, or by the slower process of starvation. The sensibility is unimpaired. The faradic contractility is at first diminished, but the muscles soon present the phenomena entitled by Erb the "reaction of degeneration." If the muscles are far advanced in atrophy, the electro-contractility may be lost. The disease in the medulla is often associated with the same degeneration in the spinal cord, when will be exhibited the phenomena of progressive muscular atrophy. Paralyses of muscles of the trunk and extremities, with contractions and without atrophy, have been observed, but these are probably complications.

Course, Duration, and Termination.—The course of the disease is progressive ; from small beginnings it grows into a formidable malady. Sometimes a stay in the progress has been noted, but only for a brief period, the course being resumed with the former intensity. The termination is fatal in from one to five years, in the mode above mentioned. An intercurrent malady may fortunately take life earlier ; pneumonia is the most usual. The frequent complication of progressive muscular atrophy, the identity of the muscular condition, and of the morbid process in the spinal cord, have led to the view, now generally accepted, that the diseases are the same, though differing as to the locality in the spinal cord affected.

Diagnosis.—Diseases of the bulb can hardly be confounded with those of other localities, because of the peculiar functional disturbances which indicate at once the seat of the mischief. Differentiation is to be made between progressive bulbar paralysis and occlusion of the vessels, acute inflammation, and tumor. Occlusion of the vessels and inflammation occur suddenly with very severe symptoms, often apoplectic, and terminate in a few days. Such is not the behavior of progressive bulbar paralysis. Tumor of the medulla and pons comes on slowly: there are, at first, symptoms of irritation, followed by depression; in progressive paralysis, the onset is slow and obscure, but there are no symptoms of irritation, those of depression occurring at once. In the case of tumor, pressure on the cavernous sinus is exhibited in swelling of the retinal veins and "choked disks," in puffiness of the eyelids and distention of the facial vein—symptoms which do not occur in bulbar paralysis.

Treatment.—Cheadle * reports a cure by the free administration of iodide of potassium, but this must have been a case of gummata. Iodide of potassium has never arrested the progress of, much less cured, a genuine case. Galvanism is the most promising remedy. Stabile applications, the electrodes on the mastoid processes, and in the opposite direction, galvanization of the sympathetic, and applications to the lips, tongue, and fauces, should be persistently used. The current should have sufficient tension to cause slight giddiness and faint flashes of light. The *séances* should be short but daily, and, if suspended occasionally, can be kept up for the necessary period. Hydrotherapy is, next to electricity, the most useful remedy. A wet pack can be worn about the neck every night, and a hot douche may be directed to the nucha for five minutes daily, but, better, a sponge dipped in hot water and kept in contact with the back of the neck for a few minutes. The good effects of the water applications are increased by the daily use of a mustard-plaster, in contact long enough to induce a little redness and nothing more. The internal medicines have not effected any improvement in the cases thus far treated. As, under analogous conditions, the chloride of gold and sodium has been of great service, it should be given a fair trial. Bichloride of mercury acts similarly. The utility of these agents probably consists in their power to check the overproduction of connective tissue. As lead and other metals, slowly introduced into the system, will produce analogous symptoms, and as syphilis has the same effect, it is good practice in every case of progressive bulbar paralysis to give iodide of potassium, freely at first—its subsequent administration being governed by the results of the first trial. From the beginning the utmost attention should be given to the diet, so as to postpone the period of decline. Soft solids are more easily swallowed, when the palate is paralyzed, than liquids. Rectal alimentation should be resorted to when the difficulty of swallowing becomes great. The injection of defibrinated blood may be employed with advantage.

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* "St. George's Hospital Reports," vol. v, p. 123.

DISEASES OF THE SPINAL MENINGES AND CORD.

HYPERÆMIA.

Definition.—As the vascular supply to the meninges and cord is the same, and as hyperæmia occurs, necessarily in both simultaneously, the term *hyperæmia* must be understood to include the contents of the spinal canal. There may be an *active*, or arterial hyperæmia ; and *passive*, or venous hyperæmia.

Causes.—Hyperæmia is the first stage in the inflammatory affections, and is a notable element in variola, typhoid, and intermittent fever. It is caused by over-stimulation of the cord in the performance of its functions : for example, protracted standing or walking, excesses in coitus, etc. Certain spinal poisons cause hyperæmia, as strychnia, picrotoxine, amyl nitrite, and alcoholic excess. The arrest of such an habitual discharge as from bleeding piles, the menses, etc., diverts an excessive quantity of blood to the cord. Probably the most frequent cause is exposure of the body while in a heated and perspiring state to cold and dampness. Congestion is produced by traumatism, concussion, etc. Workmen engaged at labor in compressed air suffer from hyperæmia, due to the solution and setting free of nitrogen in the blood of the spinal canal, as Bert has shown. Venous or passive hyperæmia is caused by obstructive disease of the heart and lungs, by cirrhosis of the liver, and by tumors of the abdomen.

Pathological Anatomy.—In active hyperæmia, vessels come into view that are invisible in health, and those of larger size are enlarged, giving to the meninges and cord a distinctly congested appearance. On section, there are more bloody points than in health ; and numerous points of extravasation, due to the rupture of capillary vessels, are to be seen. The spinal fluid is increased in amount, and is more or less reddish from the admixture of blood. Passive congestion is much more distinct, owing to the large size and numerous anastomoses of the vessels, which are greatly distended, more or less tortuous, and cause a bluish discoloration by the increase in size of the numerous small veins. Ecchymoses may also form in passive congestion, and the spinal fluid is somewhat increased in quantity.

Symptoms.—The symptoms are of two kinds ; those of irritation and those of depression. The onset is sudden in the active form, somewhat more slow in the passive form. Pain in the back, in the dorsal or lumbar region, or both, radiates downward through hips and thighs, and is increased by movements and by percussion of the skin. The pain is rather dull and heavy than acute. Pains are felt in the lower limbs, often of an acute character, and with the pain an unpleasant tingling. The skin of the lower limbs is abnormally sensitive, and the reflex excitability of the cord is somewhat augmented. A slight and usually transient sense of constriction of the abdomen is felt, and the abdominal muscles and those of the extremities are abnormally tense and rigid. There is also increased tenderness of the muscles to pressure, and they feel sore and ache a good deal, even when at rest. The electro-contractility is more prompt than in health. These symptoms of irritation occur to both forms of congestion, but they are more acute in the active form. The symptoms of depression immediately succeed those of excitation. Sensation is diminished; the lower limbs feel benumbed and heavy, and the movements are weak.

Course, Duration, and Termination.—The symptoms of irritation exist in the active form but a few hours, when the stage of depression comes on, the two groups of symptoms intermingling. The whole duration of the active form may be a few hours to two or three days. The cause continuing in operation, the symptoms will continue; but congestion can not long exist in the active form without setting up myelitis. The stage of depression coincides with the escape of fluid from the vessels and the occurrence of ecchymoses. Then the cord and the nerve-trunks being impinged on, they are functionally depressed. The termination is in recovery, if the cause is removed, or in myelitis. The onset of the passive form and the development of its symptoms are gradual; the symptoms are not so pronounced as are those of the active form, and the duration is only limited by that of the cause producing it. With various fluctuation the passive form may last an indefinite period.

Diagnosis.—Hyperæmia is distinguished from the more severe affections of the cord by the mildness and transitory character of the symptoms. From myelitis it is differentiated by the absence of fever, severe pains, contractions, paralyses, bed-sores; from meningitis, by fever, the severe symptoms of excitation and of depression; from spinal hæmorrhage, by the suddenness of the latter, and the occurrence of depression without symptoms of excitation; from anæmia, by the symptoms of general and local depression characteristic of the latter.

Treatment.—Lying on the back should be avoided. Cups or leeches to the spine, if the patient is plethoric, should be applied. If the attack has succeeded to sudden arrest of the perspiration, pilocarpine should be used to reëxcite the sweat. If the congestion is active, the spinal ice-bag may be applied. The blood-pressure should be reduced by an active purgative. A descending stabile galvanic current should be used once daily if the symptoms persist. A hot douche to the spine, every four hours, the author has found remarkably beneficial. The internal remedies most useful are, for the active form, tincture of aconite-root (two drops every two hours), and infusion of digitalis (a half-ounce every four hours), unless the symptoms of depression increase. In the active form, the author has had excellent results from the fluid extract of gelsemium (five drops every four hours); in the passive form, digitalis and ergot (j—ij ʒ fluid extract of ergot every four hours) are the most efficient means. In all cases the cause must, if possible, be removed.

SPINAL MENINGEAL HÆMORRHAGE.

Pathogeny.—Injuries and diseases of the vertebræ, penetrating wounds, rupture of a vessel from strong muscular effort, as in convulsions, tetanus, lifting a heavy weight, and the spontaneous bleeding occurring in hæmorrhagic and infectious diseases, as hæmophilia, scurvy, purpura, variola, typhoid, etc., are regarded as the causes. The most frequent position of the hæmorrhage is in the extra-meningeal connective tissue. It may form a clot entirely enveloping the dura, or occur at isolated spots, or extend over a part of the membrane. The dura itself may contain numerous ecchymoses. The coagulum may also coat the nerve-trunks up to their point of emergence. In the subarachnoid space there may be a quantity of blood, partly fluid and partly coagulated, usually quite widely distributed. In the meshes of the pia mater, or rather in the subarachnoid cellular tissue, there are layers of dark blood, partly fluid, surrounding the cord, and extending longitudinally the distance of two or three vertebræ. The cord will be compressed if the hæmorrhage is large, the part next the blood stained red and softened by imbibition. If the nerve-roots are long in contact with blood-clot, they will become stained and softened. The spinal fluid will be red, and contain particles of clot floating in it. Hyperplasia of the connective tissue, adhesions between the membranes, and extensive pigment deposits, are the results of the final changes wrought by hæmorrhage. Spinal hæmorrhage is not unfrequently associated with, or rather results from, cerebral hæmorrhage, the blood flowing down into the spinal canal.

Symptoms.—The usual onset is sudden : intense pains in the back and down the limbs are experienced, and the patient falls powerless. The other and much less common mode of onset is slower : there are pains, strange sensations, headache, and gradual failure of the lower limbs. In rare cases cerebral and spinal hæmorrhage occur simultaneously ; there are then sudden loss of consciousness, defects of speech, and syncope, in addition to the spinal symptoms. When the immediate effects of the hæmorrhage subside—the phenomena of shock, or apoplexy—then are seen the symptoms of excitation due to the presence of the blood. Intense pain in the spine about the site of the clot—the whole length, one division, or one or two vertebræ of the spine—and radiating along the peripheral tracks of the nerves impinged on in the canal. In the lower extremities will be felt the referred sensations produced by pressure on the cord—tingling, burning pain, mixed with numbness. Pressure on the motor nerves produces the signs of irritation in the muscles, chiefly contraction, rigidity, and cramp; but there may be trembling, local convulsive movements, etc. The muscles of the spine are rigid, and motions of bending or turning the body are painful. The symptoms of irritation soon yield to those of depression. Numbness, formication, diminished tactile, and painful sensations, succeed to the pain and burning; the muscles become weak, and a sense of exhaustion is experienced. Paresis of the bladder and rectum is observed when the position of the hæmorrhage is low down. In the symptomatology it has thus far been assumed that the hæmorrhage was not higher than the dorsal region. Special symptoms are produced by hæmorrhage in the cilio-spinal region, and the more if high enough to affect the origin of the phrenic. The occiput, the shoulders, and arms, are attacked by pain, spasm, and paralysis, the pupil is dilated (irritation), the respiration embarrassed (dyspnœa), there is difficulty in swallowing, and the pulse is slow and weak.

Course, Duration, and Termination.—The course of the disease varies with the site and extent of the hæmorrhage and the complications. The first stage (apoplectic) is but a few hours in duration, the stage of irritation a few days, and of depression two or three weeks. If the hæmorrhage be large, cervical, and cranial, death may ensue in the apoplectic coma; if cervical, death may be caused at once, or in a day or two, by the disturbance in the respiration and heart. Most of the cases in the dorsal and lumbar part get well, the clot being gradually absorbed. During the stage of irritation there is more or less reactive inflammation, and the products of this help to increase the after-depression. The whole course of a case of spinal hæmorrhage may be completed in one or two months, and health restored after a convalescence requiring two months. The prognosis will be influenced by the violence of the initial symptoms, by the extent of the hæmorrhage, the number and severity of the signs of irritation, and by the extent of the symptoms of depression.

Diagnosis.—Spinal hæmorrhage is to be differentiated from hyperæmia, spinal meningitis, hæmorrhage into the cord, and myelitis. It is distinguished from hyperæmia by the suddenness, the violence, and the range of the symptoms; from meningitis and myelitis, by the absence of fever, and by the suddenness of onset and more manageable character; from hæmorrhage into the cord, by the fact that in the latter there are sudden paralysis without excitation, and extensive anæsthesia.

Treatment.—Absolute quiet, the decubitus on the side or face, are the first measures. Severe pain must be combated by the hypodermatic injection of morphine, which is furthermore very useful to remove restlessness. If the hæmorrhage is going on, ergotin should be freely used hypodermatically, and general bleeding practiced if the subject is plethoric. Bloodletting is improper if the hæmorrhage has stopped. To promote absorption, the best measures are purgatives, infusion of digitalis, and the occasional administration of pilocarpine. Good results are obtained by the persistent use of ammonia—ten grains of the carbonate in a tablespoonful of the liquor ammonii acetatis three times a day. The products of inflammation (reactive) are best removed by the galvanic current to the spine daily, by the hot spinal douche, and by the spinal pack worn for a few hours at a time.

INFLAMMATION OF THE SPINAL DURA MATER—PACHYMENINGITIS SPINALIS—PACHYMENINGITIS SPINALIS INTERNA.

Definition.—*Inflammation of the spinal dura mater* corresponds to the same process of the cerebral dura mater, and the same nomenclature is used. *Pachymeningitis spinalis* means inflammation of the spinal dura mater, and it may be *external* or *internal*, the former associated with external diseases and injuries—the latter arising from ordinary causes. As the latter possesses the greater interest and importance, it is alone considered here. There are two forms of pachymeningitis spinalis interna: the hypertrophic, and the pseudo-membranous.

Pathogeny and Symptoms.—Exposure to cold and dampness combined and living in damp habitations are said to be the chief causes of the variety known as the hypertrophic. The hæmorrhagic form is precisely the same as the hæmatoma of the cerebral dura mater, and is usually found in the subjects of dementia paralytica and of alcoholic excess. In the *hypertrophic* form a great quantity of exudation is poured out on the inner surface, which solidifies into a compact connective tissue, arranged in concentric layers. This ring of indurated tissue more or less tightly embraces the cord and sets up a secondary myelitis, and, equally compressing the nerve-roots, causes them to undergo an atrophy, and the muscles to which the nerves are distributed also waste in the usual way of muscular atrophy. In the *hæmorrhagic* form a membranous exudation also takes place, developed from the sub-epithelial layer (Rindfleisch). This neo-membrane is abundantly supplied with large, thin-walled vessels, which yielding a large hæmorrhagic extravasation, in the interstices of the membrane, a cyst is thus formed, as has been described in connection with cerebral pachymeningitis. The *cervical hypertrophic pachymeningitis* is one of the numerous contributions to knowledge made by Professor Charcot, who has shown that the neck is a favorite seat of the hypertrophic form. He has shown that the first stage is that of irritation, and it coincides doubtless with the stage of membranous exudation. This first stage is characterized by violent pains in the head, neck, shoulders, and arms—pains that are continuous, and also subject to exacerbations—and are associated with a painful sense of constriction around the upper thorax. This stage of irritation continues two or three months, and is succeeded by depression. Then ensue paralysis with contraction of the upper limbs, and atrophic degeneration of the muscles, which lose their electro-contractility as regards the faradic current. Subsequently the lower limbs may become similarly affected, but to a much less extent. After remaining stationary for a long time, a change for the better may take place and a cure ultimately result.

SPINAL MENINGITIS—LEPTOMENINGITIS SPINALIS.

Definition.—When the term *spinal meningitis* is used it is intended to express inflammation of the arachnoid and pia mater, for no distinction between the two is possible either in respect to the pathological or clinical standpoint. There may be an *acute* or *chronic* form.

Causes.—It is a disease of the male sex, and occurs in youth and adult manhood. All depressing influences and the evils of bad hygiene tend to develop it, and it attacks by preference the subjects of the scrofulous cachexia. Exposure to cold and dampness, while the body is warm and perspiring, is an influential factor. Penetrating wounds and injuries and diseases of the vertebræ have a direct effect which is unquestionable. Neighboring diseases affect the spinal meninges by contiguity; those of the brain have the most immediate connection. It occurs also during the course of acute infectious diseases, as puerperal fever.

Pathological Anatomy.—After an intense hyperæmia of the membranes, punctuated by ecchymoses, much fluid is exuded, and the tissues are swollen and infiltrated with serum. A quantity of exudation partly purulent and partly fibrinous is poured out; the spinal fluid becomes reddish and muddy from the presence of cells, flakes of fibrin and pus; the membranes are infiltrated with pus-cells, and are coated more or less extensively with patches of fibrin, the whole length of the cord nearly being covered with exudation. The roots of the spinal nerves are also thickly covered with exudation and bathed with a pathological fluid—the result is, they are swollen, softened, and more or less injured by imbibition. The cord itself never escapes entirely; it may be only sodden; it may be softened, congested, and œdematous. In the chronic form there may be adhesions of the membranes, pigmentation, large accumulation of fluid, atrophic and sclerotic degeneration of the cord, etc.

Symptoms.—There may or may not be a chill to mark the onset of the disease, but a rise of temperature, general *malaise*, headache, nausea, and constipation, with the urine acid and high-colored, indicate the beginning of an inflammatory affection. Then occur the local pains, which attract attention to the spine—pain, of a severe, deep, boring character, in the loins, back, or neck, usually in the dorso-lumbar region, rigidity of the spine, a constriction or girdle of severe pain around the body, and pains radiating downward into the limbs. The motor nerves excited by the exudation cause the muscles to which they are distributed to assume a state of spasmodic contraction, limited to the lower limbs, to the rectum and bladder (retention of urine and constipation), when the lesions do not extend above the last dorsal; extending to the muscles of the trunk and the superior extremities, to the respiratory and posterior cervical muscles, if the cervical portion of the meninges is invaded. When this portion of the spinal canal is occupied by the inflammation, there occur dysphagia, dyspnœa, slowing of the pulse, and feebleness of the heart. Striking on the spinal processes does not necessarily awaken pain, but much soreness is felt when the spine is bent in the movement of the body. It is important to note that the muscular contractions are excited and increased by all attempts at movement, whereas irritation of the skin does not have this effect—a point of differentiation between meningitis and tetanus (Jaccoud). With this condition of the motor functions, there are also hyperæsthesia and hyperalgesia of the integument in the area of motor derangement. When the respiratory muscles are affected, at this stage death occurs early, the pulse becomes very rapid, the dyspnœa increases and asphyxia results. Otherwise, the acute symptoms subside, and the remission may be the beginning of convalescence. More often this diminution of the acuity of the symptoms and the moderation of the excitation denote the onset of the paralytic—the stage of depression. The paraplegia is not complete; partial contractions remain in the paralyzed members, and more or less hyperæsthesia persists. Constipation from paresis and urinary retention are replaced by incontinence, but this is not invariable. Reflex movements are not abolished. Anæsthesia will more or less, but not entirely, replace hyperæsthesia. The electro-contractility (faradic current) is not impaired in some muscles, but is weakened and lost in others. The extensors are more often affected by atrophy and loss of electro-contractility (Rosenthal *). The cases may now follow two courses: In one the symptoms of paralysis will invade the respiratory muscles, and death will occur in coma (carbonic-acid poisoning), the temperature sometimes rising to an extraordinary height. In the other case, the course will be more protracted; there will be periods of apparent improvement, but the paralysis will extend, bed-sores will form, urine will dribble away, and death occur finally by exhaustion. If the disease extend to the medulla, there will be produced, besides the disturbances of respiration and of the heart which occur when the cervical meninges are inflamed, affections of speech, vomiting, ocular derangements, delirium, etc. There is no characteristic thermal line ; the fever is high at the outset, but the temperature declines during the stage of depression, to rise sometimes to an extraordinary height during the death-agony. The appetite is lost, the body wastes rapidly, and emaciation, in the cases with bed-sores and death by exhaustion, proceeds to a remarkable extent. The *chronic form of spinal meningitis* succeeds to the acute cases of moderate severity, or originates spontaneously—the latter more frequently. It presents the same form and order of symptoms—those of excitation, those of depression. These effects are due to effusions and membranous exudations in the spinal canal. The membranes are thickened, pigmented, and adherent to each other and to the cord. The pressure of the contracting sclerotic connective tissue induces atrophy of the nerve-roots, and if the posterior roots are impinged on degeneration may occur in the posterior columns (Rosenthal). The cord itself is ultimately damaged by a parenchymatous myelitis. The symptoms of irritation are chiefly expressed in disorders of sensibility, muscular rigidity and spasm being partial and fugitive. The pain is felt in the lumbar region and through the lower limbs, and has a rheumatismal character. The pain is accompanied by hyperæsthesia, which, however, is never so considerable as in the acute form. Paraplegia develops slowly : at the first there is a strong sense of fatigue, then of increasing weakness ; numbness, tingling, and slowly marching plantar anæsthesia, come on in the order named. The weakness extends to all the muscles of the inferior extremity, and to the rectum and bladder, and may ultimately invade the upper extremities, always in its march attacking the two sides of the body equally. This form of paraplegia is irregular in its progress—now advancing, now receding.

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* "Klinik der Nervenkrankheiten," Stuttgart, 1875, p. 286.

Course, Duration, and Termination.—The fulminant form terminates in a few hours or a few days, its course being characterized by the extent and diffusion of the symptoms, the early implication of the cervical portion, and consequent failure of the lungs and heart. The ordinary severe form lasts two or three weeks, and terminates in either of two modes : in from one to two weeks by the embarrassment of respiration and weakness of the heart, coma developing in consequence of carbonic-acid poisoning ; in from two to four weeks, by gradual failure, death being due to exhaustion. The severe form may terminate in recovery. At the end of the excitation period a remission in the symptoms occurs, the stage of depression does not develop into paraplegia, and convalescence proceeds slowly, the health being reëstablished not until two or three months have elapsed. In the most favorable cases a change for the better may take place in the excitation period in a few days, and convalescence be established, or the symptoms be resumed in a milder form, convalescence being then established. Not unfrequently some critical evacuation, such as a profuse sweat or urinary discharge, an epistaxis, or menstrual or hæmorrhoidal discharge, marks the cessation of the morbid process, and a rapid recovery then takes place. More frequently the recovery is slow, owing to extensive exudations, and there is a long period of lameness or paralysis. Again, recovery may ensue with permanent disability of a member, or group of muscles. In any case, the prognosis is serious.

Diagnosis.—The distinction between tetanus and spinal meningitis rests on these points : trismus is among the first symptoms of tetanus, and rarely occurs, and then later in spinal meningitis ; risus sardonicus is peculiar to tetanus; the spasms are rhythmical in tetanus, are more severe, and are excited by reflex causes—similar spasms do not occur in meningitis, are much less severe, and are only excited by movements. In tetanus, no oculo-pupillary phenomena, no changes in the cranial nerves, no delirium, no fever—all occur in meningitis. The history of the case, especially the presence of a wound, will often decide. From myelitis, meningitis is differentiated by the pain in the back, the hyperæsthesia, the muscular rigidity, and on the part of myelitis by the early paraplegia and anæsthesia. Rosenthal places much stress on the electrical state of the muscles—the electro-contractility and sensibility (faradism) of the nerves are much lessened, or disappear entirely in spinal meningitis. From typhoid fever, by the thermal line, by the absence of the irritation symptoms, by the diarrhœa, by the stupor—in fact, the least attention ought to decide promptly.

Treatment.—Absolute repose in a darkened room, the decubitus lateral or on the face, must be insisted on. Leeches or cups to the spine during the period of excitation—the amount of blood drawn being dependent on the vigor of the subject. The application of the spinal ice-bag may be proper, but caution is necessary. The author has a strong conviction that hardly any topical application is to be compared with the hot douche to the spine, or, instead, a large sponge dipped in hot water and passed frequently over the spine. The most efficient internal medicines are opium, aconite, and ergot—two drops of the tincture of aconite-root, five to ten drops of the tincture of opium (deodorized), and fifteen to thirty minims of the fluid extract of ergot every two hours during the stage of excitation. If the pain is very severe, the hypodermatic injection of morphine may be necessary at the outset. As opium is a remedy of the greatest importance, its effects should be steadily maintained during the excitation stage. When the symptoms of depression come on, quinine (three grains) and belladonna extract (one fourth of a grain), every four hours, are the most useful remedies. The paralysis of muscles during the period of convalescence is best treated by faradization, or galvanism slowly interrupted, if the former fails to induce responses. The galvanic current should be applied to the spine and to the nerve-trunks. After the acute symptoms have subsided, strychnine may be injected into the paralyzed muscles. Massage to the paralyzed members or muscular groups is an expedient of great utility. During the excitation period, and after cups or leeches have been applied, mustard-plasters to produce slight rubefaction are highly useful. Twice a day, a mustard-plaster four inches broad should be put on from the occiput to the sacrum, and removed as soon as slight redness is caused. During the stage of depression, *flying*-blisters to the spine are highly serviceable. Great circumspection is necessary, since all severe counter-irritation may help to form bed-sores. To remove deposits from the spinal canal, especially in the treatment of the chronic form of spinal meningitis, and the pachymeningitis interna of the cervical region, there is no remedy so efficient as the iodide of potassium, especially when its actions are aided by the occasional administration of pilocarpine.

ACUTE MYELITIS.

Forms.—*Myelitis* implies an inflammation of all the tissues of the cord. There are several forms, determined by the seat and range of the inflammation. It is sometimes designated *diffused* myelitis, and it may be *acute* or *chronic.* It is further distinguished into *central myelitis,* when the inflammation occurs chiefly in the central gray matter; into *transverse myelitis,* when all the tissues of the cord at certain levels are involved, as, for example, *dorso-lumbar*, *dorsal*, and *cervical* transverse myelitis; into *hemilateral*, when a longitudinal half of the cord is affected, and *disseminated*, when there are spots of inflammation scattered along the cord at various points through its whole length.

Causes.—Myelitis is more common in males than in females; in youth and early manhood than in advanced life. One form occurs in childhood. Contusions, blows, fractures of the vertebra, severe and prolonged functional activity of the cord, as in protracted standing, excesses in coitus, self-abuse, exposure to cold and dampness combined, are the most common causes. Inflammation of the cord may be excited by neighboring inflammations, transmitted by contiguity: meningitis, traumatic inflammation of the dura, and carcinoma, are the representatives of this group of causes. It is one of the complications of typhus, the exanthemata, puerperal fever, and acute rheumatism. The so-called reflex paraplegias are often, probably, examples of myelitis.

Pathological Anatomy.—The first step in the process is hyperæmia, which is usually very intense, the affected area being deeply red. Extravasations also occur, and hence the tissues may have a reddish-brown or chocolate tint. With the hyperæmia occur serous transudations, so that the inflamed district is moist and juicy, and softened. A change in coloration next takes place to yellow, and ultimately to white, the nerve-elements are disassociated, become fatty, and finally an emulsioned mass remains, of creamy appearance and consistence. The meninges of this part of the cord take part in the inflammation, become thickened, opaque, and infiltrated with pus-cells, and contract adhesions. Such are the macroscopic or naked-eye appearances. On microscopic examination the changes consist in dilatation of the capillaries, arterioles, and veins; in the migration of the white and diapedesis of the red corpuscles; in fatty and granular infiltration of the walls of the vessels; in the exudation of a colloidal hyaline substance about the vessels; in swelling and proliferation of the neuroglia-cells, and a hyperplasia of the reticulum; in the exudation in great numbers of granule-cells in the interstices; in the granular disintegration of the nerve-fibers, the axis-cylinders forming ampullary dilatations: and in swelling, proliferation and granular atrophy of the ganglion-cells. The continued development of these morbid processes results in the almost entire disappearance of the proper elements, the remaining mass being composed of fat-granules, hypertrophied neuroglia, dilated and thickened vessels. Cysts are sometimes seen, composed of a dense connective-tissue envelope, and a reticulum of the same, containing serum and detritus. Without proceeding so far as the complete destruction of the nerve-elements (cells and fibers), which is the ultimate step in the acute process, a transition to the chronic forms is effected, in which there is an hyperplasia of the neuroglia, the spider-cells enlarge and increase in number, the vessels undergo thickening, numerous amylaceous corpuscles or bodies appear, while the nerve-elements atrophy. The central gray matter is the chief seat of this disease, but it extends so as to involve all parts. It may be most severe in the gray matter; it may have an hæmorrhagic character, and it may consist chiefly in a hyperplasia of the neuroglia.

Symptoms.—The usual course is the onset by a chill, fever, and general *malaise*. Or the spinal symptoms begin without any preliminary. There are experienced intense pain in the back, with a band of pain and constriction around the body, soreness developed by percussion of the spine, pains and muscular soreness of the limbs, tingling, formication, a feeling of weight and dragging in the rectum and bladder, and priapism. There may be, but not invariably, corresponding symptoms of irritation in the motor sphere, such as tremors, spasmodic contractions, clonic convulsions partial, even general. But paralytic symptoms appear in a few hours, and soon complete paralysis, and disappearance of the electro-contractility (reactions of degeneration). Paralysis of the sensory nerves also takes place in a short time, and sensation is lost more or less completely in all the affected region up to the upper line, often terminating quite abruptly about the middle of the body. The analgetic parts may also be the seat of violent pains (*anæsthesia dolorosa*). Paralysis of the sphincters may follow very soon the paralysis of the muscles, but it may be delayed for some time, and in other cases it may not occur at all. The condition of the reflex function varies greatly. All reflex activity may be abolished ; it may be diminished ; it may be unchanged ; it may be greatly exaggerated—the variations being due to the position and extent of the lesion in the cord. Sometimes the paralysis reaches its highest at once and is afterward stationary ; sometimes it ascends the cord and rapidly involves the parts above ; sometimes the extension is transversely, all parts of the cord in turn being affected. When the inflammation extends horizontally and affects the anterior cornua, the paralyzed muscles waste rapidly, and bed-sores form quickly and spread widely. These *trophic* lesions also excite disease of the mucous membrane of the genito-urinary tract, the urine becomes alkaline, and a violent and destructive pyelonephritis and cystitis are set up, the paralyzed limbs become œdematous, and effusion takes place into the joints. If the myelitis is of the ascending variety, when the ciliospinal region is reached, pupillary phenomena are observed—enlarged pupil, if the sympathetic centers are merely irritated ; contracted pupil, if these centers are destroyed. When the cervical portion of the cord is reached, the muscles of respiration becoming paralyzed—the intercostals and trunk-muscles—breathing can be carried on only with the diaphragm, and finally, this muscle being paralyzed, there are most intense dyspnœa, rapid filling of the lungs, and death. The fever with which many cases are inaugurated pursues no defined plan. In some cases fever persists throughout, in many it is paroxysmal, but without regularity, in others it does not appear at all. In some instances intense fever precedes death, and is higher than ever immediately after death. The pulse is frequent usually, very frequent and irregular when the cervical portion of the cord is invaded. The nutrition in some cases fails rapidly, in others is preserved fairly well. There are obstinate constipation and meteorism produced by paralysis of the muscular layer of the bowel.

Course, Duration, and Termination.—There are numerous variations in the course of the disease, due to the position and tendency of the lesions. If the paralysis is of the ascending variety, the respiratory muscles soon become involved, and death takes place in a few days by asphyxia. In other cases, the trophic center being invaded, there occur extensive bed-sores, intense pyelonephritis and cystitis, changes in the joints, and death by exhaustion in three or four weeks, or as many months. It occasionally happens that the morbid process is arrested at a certain stage, and the health is restored ; but, permanent damage having been inflicted, permanent deformity remains, such as wasted and paralyzed muscles, contractions, and deformities of joints. In still other cases, the acute passes into the chronic form of the disease. Rarely, complete recovery ensues. When this result takes place, a remission occurs at an early period, the paralysis is not complete, and slow absorption of exudations is effected. The myelitis from traumatic causes is usually situated above the dorso-lumbar enlargement, and is of the variety known as *myelitis transversa*. The symptoms present are the constricting band around the body, spinal pain, paraplegia, anæsthesia, no atrophy of the muscles, paralysis of the bladder, and reflex contraction of the muscles more active than normal. The electro-contractility of the leg-muscles is preserved. Central myelitis affects the gray matter, including the anterior horn. This form begins abruptly, proceeds rapidly, and involves sensation and motion and the trophic functions. The reflex excitability and the electro-contractility (faradism) are quickly extinguished (reactions of degeneration), the muscles waste rapidly, the muscles of respiration are quickly paralyzed by extension upward of the disease, and death occurs early by asphyxia. The hæmorrhagic form differs from the purely central myelitis by the still more abrupt appearance of the paralysis.

Diagnosis.—Myelitis may be readily confounded with meningitis: they differ especially in respect to the stage of irritation, which is pronounced in meningitis, but hardly recognizable in myelitis. In meningitis, there are rigidity, spasms and contractions of muscles, pain and hyperæsthesia; in myelitis, paralysis appears in a short time, involves the rectum and bladder, and anæsthesia follows. The electro-contractility is preserved in meningitis, but often lost in myelitis. Hæmorrhage in the spinal canal is distinguished by its abruptness, the irritative symptoms (absent in myelitis), the slight paralysis and preservation of electro-contractility, as against the severe paralysis, wasting of muscles, loss of reflex and electric excitability, and trophic disorders characteristic of myelitis. Hæmorrhage into the cord is recognized by the abruptness of the symptoms, sudden paralysis without fever or other constitutional disturbance, the loss of power being stationary.

Treatment.—Absolute rest and the avoidance of all excitement, decubitus on the side or face, and careful and nutritious alimentation, are the first measures. The frequent application of hot water to the spine—preferably the hot douche—is very serviceable; in some interval between these applications, a mustard-plaster the length of the spine and four inches broad should be put on, and retained no longer than beginning rubefaction, and repeated twice a day. Internally, the best results are obtained from the infusion of digitalis (a tablespoonful four times a day), for this remedy is preferable to ergot in the acute inflammatory affections of the spine, owing to the peculiar arrangement of the spinal veins. Local bloodletting and purgatives are useful in plethoric subjects. As the stage of congestion passes into the stage of exudation, ammonia (the carbonate) should be given freely (five grains every three hours). Any specific infection must be regarded in the plan of treatment adopted. As the stage of depression develops, quinine in small doses, and belladonna extract, may be very useful. Scruple to half-drachm doses of quinine may have a good effect at the very beginning of the congestion stage.

CHRONIC MYELITIS.

Forms.—Under the term *chronic myelitis* are included various changes in the cord, of induration or sclerosis, and gray or gelatiniform degeneration, and, less often, of softening. The several forms of chronic myelitis, named according to their seat and character, are *central, transverse, progressive,* and *disseminated myelitis.* Each form has its distinctive symptomatology, because of the functions of the different parts of the cord.

Causes.—The causes of chronic are much the same as those of acute myelitis. It may arise from the acute form; may be due to injuries, concussions, blows on the spine; may result from sexual excess, from exposure to cold and dampness, or from the arrest of some habitual discharge. The so-called reflex paraplegias are probably nothing more than chronic myelitis, arising from reflex disturbances.

Pathological Anatomy.—The changes are of several kinds. Macroscopically there may be no alteration, or the consistence and color may be visibly changed. As to consistence, there may be sclerosis or softening, the latter much less frequently, and in color the change is to a grayish or yellowish-gray discoloration—an evidence of the existence of gray degeneration. The patches of sclerosis may be localized, or diffused, or disseminated. The changes may be limited to the central gray matter, and especially to that part surrounding the central canal, or to the gray matter of the anterior cornu, or to the lateral columns or to the posterior columns. Again, the peripheral part of the cord may be affected in conjunction with the pia.* The nerve-roots may be more or less advanced in the gray or gelatiniform degeneration, the nerve-trunks atrophied, and the muscles to which they are distributed equally affected by an atrophic degeneration, partly fatty. Various trophic changes occur in the joints and mucous membrane of the genito-urinary tract, and bed-sores form. The microscopic changes consist in an hyperplasia of the neuroglia—the fibers increase in number and size, and the cells undergo a nuclear proliferation. Various changes occur in the nerve-fibers: they may be swollen, disintegrating, fatty; the axis-cylinder equally atrophied or indurated. The ganglion-cells are shrunken, pigmented, indurated, lose their processes, and their nucleus and nucleolus alike disappear. The vessels also undergo important changes: the adventitia is indurated, and is the seat of nuclear proliferations and formation of fat-cells, and is thickened as well as indurated. Numerous fat granules and cells and corpora amylacea are distributed through the sclerosed patches.

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* Vulpian, "Archives de Physiologie," tome ii, p. 279, "Note sur un cas de méningite spinal et de sclérose corticale annulaire de la moelle épinière."

Symptoms.—The symptoms are at first without much significance. Disorders of sensation usually precede the motor disturbances. There are pains in the limbs that have the character of and are usually confounded with muscular rheumatism, tingling, mixed with numbness, and some burning; pain in the back, and a sense of constriction around the body—the girdle or band feeling; sometimes the integument over the spine is highly sensitive. Motor disturbances next appear. Muscular fatigue is felt without exercise, and becomes severe when any effort, as in walking, is made. The feet and legs feel heavy, and their movements are awkward. With the progress of the case, sensory depression, after a time, supersedes all the symptoms of excitation. Numbness is felt in the fingers in the distribution of the ulnar nerve, in the toes, and in the bottoms of the feet, which feel as if a cushion were interposed between them and the floor. The various endowments of the sensory nerves disappear in turn—first the impression of tickling, then touch, pressure, temperature, and finally pain (Rosenthal). The anæsthetic area is the front part of the thighs, the hips and loins, the inferior portion of the body upward to either side of the abdomen. There are parts below the girdle-line in which sensation is only lessened, and parts that still retain their normal sensibility. Strange aberrations of sensations are observed in the anæsthetic regions—the application of heat may cause a sensation of coldness, of cold, a hot or burning feeling. Furthermore, an impression made at any spot may be referred by the patient to some distant point, or indeed to the other side of the body. The rate at which impressions are transmitted from the periphery to the centers of consciousness is much lessened in this disease owing to the obstacles in the paths of conduction—seconds even being occupied in the passage of an impression from the great-toe to the sensorium. The paresis or paralysis extends from below upward, very rarely in the opposite direction. The position of the paralysis depends on the part of the cord invaded. If the cervical portion, the upper extremities will be the seat of motor and sensory disorders, the pupils will be unequal, there will be embarrassment of respiration in consequence of paralysis of the intercostals and muscles of the chest above, the action of the heart will be rapid and weak, there will be suffocative attacks, and difficulty in swallowing. If the dorso-lumbar enlargement be involved, there will be the paralysis of the lower limbs (paraplegia), of the bladder and rectum, the electro-contractility (reactions of degeneration) and the reflex excitability will be both abolished; but, if above the dorso-lumbar enlargement, the reflex and electro-contractility will be rather heightened. The paralyzed muscles waste and lose their electric reaction—the anodal disappearing before the cathodal reaction. The sexual functions decline correspondingly. At first there is priapism, but the erections presently cease altogether; yet nocturnal pollutions occur from time to time until absolute impotence results. The urine is at first frequently discharged with difficulty; there may be incontinence and dribbling, or retention and a catheter needed. Constipation and meteorism are present, because the muscular layer of the bowel is either paretic or paralyzed. The general nutrition often continues in a satisfactory state throughout, but, in the severe cases and toward the end of most cases, much suffering is experienced from the wakefulness, bed-sores, the incontinence of urine, and the inflammatory reaction from cystitis and pyelonephritis.

Course, Duration, and Termination.—The development of the disease is slow, whether the chronic succeeds to the acute or originates *de novo*. Its progress is slow, and, although varied by periods of apparent improvement followed by exacerbations, its tendency is downward. Nevertheless, there are in many cases long periods of a perfectly unchanging state in which the damage done continues, and no change for the worse takes place for many years. Even in those cases which seem stationary, there should be not too confident hopes of an arrest, since relapses may occur. In any case there can be no true recovery; only an arrest of the morbid action, for the damage done is permanent. There are various modes of termination: by cystitis, pyelonephritis, and bed-sores, by some intercurrent malady, as pneumonia or pleuritis, or by the extension upward into the cervical region.

Diagnosis.—We have first to distinguish the several forms of myelitis, as regards the seat of the lesions and the mode of their progression. When the cervical portion of the cord is affected, the symptoms of irritation and depression are seen in the hands and arms, in the disturbances of respiration and circulation, in the oculo-pupillary phenomena, the lower extremities and the sphincters becoming affected subsequently. If the dorsal portion is affected, above the dorso-lumbar enlargement, the respiration will be affected by paralysis of the intercostals, the constricting girdle will be high up about the nipples, there will be paraplegia and paralysis of the sphincters, but reflex and electro-contractility will not be affected, rather heightened than diminished. If the lumbar region is affected in addition to the symptoms of the dorsal, there will be loss of reflex and electro-contractility and usually the trophic disorders. When the disease invades the multipolar cells of the anterior horns, it is called *poliomyelitis anterior chronica*, the paralytic symptoms occur as in the disease of the other parts of the cord, but in this region lesions produce trophic changes in the paralyzed parts, rapid wasting of the muscles, changes in the joints, bed-sores, cystitis, etc., and loss of reflex and electro-contractility. Chronic myelitis is distinguished from hæmorrhage into the cord by the suddenness of the onset, and the prompt development of paralysis characteristic of the latter. From spinal meningitis, by the excitation symptoms, and the preservation of the reflexes and the electro-contractility, and the presence of febrile excitement, all wanting in chronic myelitis.

Treatment.—If the disease is recent and advancing, rest takes the first rank as a remedial agent. The rest must be as nearly absolute as possible, and should be kept up for two to three months to be of any service. Erb regards the hydropathic method as the most successful; the local application of cold water by compresses to the spine, removed when they get warm; the "rubbing wet pack," the application restricted to the back and body, hip-baths, and the half-bath, with douches to the spine.* The temperature of the water should not exceed 80° Fahr., and should not fall below 55°, and the treatment should not be continued too long. If patients do not react well and remain chilly, the treatment does no good. The author has had remarkably good results from the application of the hot douche in cases of myelitis. Next to hydrotherapy, galvanism is the most useful agent. The important point, too little understood, is the use of a large volume and low tension. From forty to sixty elements of Siemens and Halske and large sponge electrodes well moistened are the principal needs. The individual applications should be about two to five minutes' duration, and should be made daily. The duration of the treatment will be influenced by many considerations, by the benefit or injury especially. Even if it do good, the current should not be used daily for months at a time, but a few days' intermission every month are necessary. The direction of the current seems a matter of indifference, but the author believes, if the blood-supply is to be increased and the nutrition improved, that the descending current is better. Nitrate of silver has been beneficial in many cases. The author has seen good results from the chloride of gold and sodium, alone, and in combination with minute doses ($\frac{1}{30}$ gr.) of corrosive sublimate. Of all the agents for the period of depression, the author regards the lactophosphate of lime as the most permanently beneficial. It may be given with arsenic and contemporaneously with cod-liver oil. The diet must be light and easily digested, especially so in those cases undergoing the rest-cure. Spirits must be forbidden. One of the most unpleasant complications of myelitis—incontinence of urine—may often be relieved by faradization of the bladder, which is best accomplished by introducing a button electrode into the rectum, and applying a sponge electrode to the hypogastric region.

POSTERIOR SPINAL SCLEROSIS—PROGRESSIVE LOCOMOTOR ATAXIA.

Definition.—*Posterior spinal sclerosis* is a form of myelitis, which does not extend transversely but longitudinally, and is limited to the posterior columns. The term *progressive locomotor ataxia* was applied by Duchenne to designate the special characteristics of the malady. This disease has long been known in Germany under the term *tabes dorsalis.*

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* See the author's "Materia Medica and Therapeutics," sixth ed., art. "Hydrotherapy."

Causes.—Probably the chief cause of posterior spinal sclerosis is inherited tendency. Some striking examples of this disease appearing in collateral family lines have been reported by Friedreich.[*] It is sometimes directly transmitted; thus, Carré has reported an instance of one family, among whom there were eighteen cases in three generations.[†] It is a disease of the most active period of life, occurring from twenty to sixty, but the cases are most numerous between thirty-five and fifty. It attacks males twice as often as females. Occupations involving exposure to cold and dampness, to fatigue, and depressing moral emotions, favor the development of the disease. It is alleged that railroad-engine drivers, stokers, conductors, and brakemen, suffer from this and other spinal diseases by reason of the concussion. There are no statistics or exact observations thus far published on this point. Sexual excesses are generally held to be influential in causing this disease, but, as an unusual salacity is one of the first manifestations of the changes taking place in the cord, there is danger of confounding cause and effect. There seems to be no doubt that there is a relation between rheumatism and locomotor ataxia.[‡] The author has seen a well-marked case, produced in a gilder by his occupation, the symptoms ultimately disappearing under iodide of potassium. It is probable that the slow absorption of the metals used in the arts is often responsible for the production of symptoms similar to those of posterior spinal sclerosis. That the syphilitic cachexia stands in an intimate causal relation to this disease seems well established by modern researches. By this statement, it is not intended to express the notion of an ataxic condition due to syphiloma of the spinal cord, but rather that the disease arises in the posterior columns in consequence of the development of a peculiar cachexia, the product of syphilitic infection and of the remedies used for its cure.

Pathological Anatomy.—The meninges may be unaffected, but in a majority of cases the pia mater presents the appearances of increased vascularity along the region of the posterior columns. The form, color, and consistence of the cord are altered. The change consists in an atrophy of the posterior columns, and hence there is a shortening of the antero-posterior diameter; in a gray, semi-transparent, rather vitreous, amber, rose or reddish-yellow color, which contrast strongly with the adjacent whitish nervous matter, and in an increase of the consistence of the affected area, although it may also be softer than normal. The extent of the degeneration varies in different cases, but in general it occupies the parts between the posterior roots, and is most considerable in the dorsal and upper lumbar portion of the cord, but it may extend from the filum terminale to the calamus scriptorius. The changes, microscopically studied, consist in a hyperplasia of the connective tissues, a granular degeneration, atrophy, and disappearance of the proper nerve-elements, the accumulation of fat-cells, pigment, and corpora amylacea. The posterior roots are also affected by a fibroid change—the connective tissue undergoing development, the nerve-fibers wasting. Not all parts of the posterior columns are equally affected : in the lumbar region the external division, in the cervical the inner and middle division or the columns of Goll are chiefly diseased. Similar alterations take place in the gray posterior horns, and extension of the morbid process ultimately is effected to the lateral columns. The spinal ganglia and anterior nerve-roots escape degeneration, as also the ganglia of the sympathetic system. The gray degeneration often attacks the optic nerves, sometimes the oculo-motor and the abducens. The joints undergo remarkable changes : the articular cartilages disappear by absorption, the head of the bone and the articular cavity gradually flatten, atrophy, and are greatly changed from their normal appearance.

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[*] "Ueber Ataxie mit besonderer Berücksichtigung der hereditären Formen," von Professor Dr. N. Friedreich in Heidelberg, Virchow's "Archiv," Band lxviii und lxx.

[†] Erb, *op. cit.*

[‡] Topinard, "De l'Ataxie Locomotrice," etc., Paris, 1864, p. 363.

Symptoms.—In a man of the middle period of life, apparently in good health, there appear from time to time severe pains in the body, hips, thigh, and leg. These are usually of two kinds—sharp, quick, lightning-like pains flying through the limb, and a feeling of muscular pain, which leaves a sensation of soreness. These pains at first are occasional but after a while they become paroxysmal and somewhat more frequent, and may, by the time the other symptoms are defined, be present more or less every day, although they may disappear for weeks at a time. The pains are increased by cold, especially by cold and dampness combined, and are worse in winter. At or before the onset of the pains there is a marked increase in the sexual appetite, and men are driven to commit excesses to which they had previously been strangers. The period of pains, with or without increased sexual inclination, lasts a variable period, from a few weeks to several years, and is very often diagnosticated and treated as rheumatism. These pains are most severe in those parts destined to become ataxic first, usually the lower limbs. The next symptom is *diplopia*, which appears unexpectedly and after a variable period of a few weeks or a few months, disappears as unaccountably, although the change is very often attributed to the remedies of some oculist consulted by the patient. Besides the visual disorder from this cause, the eyesight gradually becomes dim (amblyopia), and further on, the gelatiniform degeneration attacking the optic nerve, vision is lost (amaurosis). During this period the salacity, which was at first active, begins to decline and nocturnal seminal losses occur. There is also less and less ability to satisfy the desire, the sexual congress becoming unsatisfactory, the erections inadequate, the ejaculation premature, and more or less pain taking the place of the pleasurable sensations, and finally complete impotence results. The first stage, according to the definition of Duchenne, consists of three symptoms: *pains, ocular disorders, anaphrodisia.* As already remarked, the duration of this stage varies within wide limits—from a few months to several years, and then begin the symptoms characteristic of the so-called second stage: numbness; ataxia of the muscular movements of the inferior extremities; cutaneous and muscular anæsthesia. In the bottoms of the feet the numbness includes a sensation as if cotton-wool or a cushion were interposed between the feet and the floor; the constricting girdle sensation of spinal diseases is experienced around the body at different heights; the limbs, thighs especially, feel as if embraced by a tight-fitting cuirass; the severe, lightning-like pains rather increase than diminish; the sense of touch is impaired, so that the points of the æsthesiometer can be felt as two only when they are very far apart; impressions of irritation are slow to reach the centers of consciousness; the sense of pain declines and is entirely abolished, but this latter may be at particular points only; the sense of pressure and the sense of temperature are diminished. As regards the motor functions, we find the following characteristic phenomena: the "knee-jerk" or patellar tendon reflex is found to be absent; at first the limbs are easily fatigued and the movements are uncertain, so that in walking the gait has an unsteadiness like that of slight alcoholic intoxication, and these unfortunates are often suspected of indulging in this vice; a sense of insecurity and often of helplessness, as when a carriage is approaching rapidly, or walking on a marble or tiled floor, or in the obscurity of the evening, is experienced; the ataxic phenomena increase so that they can not stand with the eyes closed, and in walking the feet describe a semicircle, the toes pointing upward and outward, the heels coming down with a stamp. An examination of the muscles now discloses that the disorder of locomotion is an ataxia; the muscles are not weak at first, and very great ataxia may coexist with complete retention of muscular power, but presently some of the muscles become paretic, and ultimately there may be paralysis with wasting. They can not at first walk without the aid of vision; after a time the assistance of a cane is needed besides their eyes, then two canes are found necessary, and finally walking has to be abandoned. In Duchenne's rather arbitrary arrangement the third stage consists in the extension of the sensory and motor disturbances to the upper extremities. The order of phenomena is as follows: pains, numbness, first in the ulnar-nerve region, then extending to all the fingers, troubles of coördination, inability to use the knife and fork, to fasten a button, etc. The reflexes are variously affected—sometimes increased, sometimes lessened, sometimes wanting; but the absence of the patellar tendon reflex is one of the most characteristic signs, so very rarely is it wanting in health, and so constantly is it not found at an early period of this disease. According to Buzzard* it is absent in 96 per cent. of the cases. Electro-contractility is increased or normal, and reduced or lost when muscles degenerate.

During the progress of the case, usually the vegetative functions are well performed. The appetite remains good, and the nutrition does not fail; the patients often having a rosy countenance and a self-satisfied expression, which lends countenance to the theory of secret drinking. The mental functions continue unaffected, and the moral state is one of contentment, although there may be great depression of spirits. There are peculiarities in the symptoms, not included in the preceding description, to which some attention should be paid. The anæsthesia of the soles of the feet is an element in the disorders of movement in walking. Some patients with entire analgesia, so that a pin can be driven into the flesh without any sensation whatever, suffer agony with a gentle touch, as the brushing of a woman's dress against the legs. It is in spots that such sensations exist. The place where a lightning-pain has just been felt often burns for some time after. One of the most disagreeable disorders of sensation is the feeling of "fidgets," a peculiar unrest which impels to movement. The muscular sensibility is much reduced. The muscular sense, the knowledge of the position of the members, and the appreciation of weight and resistance, are all reduced or abolished, and consequently the disorders of muscular action of every kind are enhanced. The ataxia of movement is particularly well exhibited when the patient, lying recumbent, is told to touch objects with his foot. The movements are in jerks, great energy is put into them, but the direction is irregular and apparently purposeless. Ataxia affects the muscles of the eye, as well as of the extremities, producing the effect called nystagmus, disordered accommodation, and changes in the size of the pupils. Friedreich's bilateral nystagmus consists of jactitating movements in a vertical, horizontal, or oblique direction, not when the eye is at rest, but when an attempt is made to fix it. Besides these motor disturbances, vision is affected by gelatiniform degeneration of the optic nerve, in a variety of ways—in respect to the size and sharpness of the field of vision and the appreciation of colors, the ultimate result being white atrophy of the optic disks. Various trophic alterations occur during the course of locomotor ataxia, especially toward the end. The most important, which has already been referred to, are the joint affections, beginning usually in the knee-joint. These changes may indeed begin before the ataxia, during the first stage, and involve the shoulder, elbow, and wrist, as well as the knee and hip. There occurs first, in the joint, swelling due not to any inflammatory process, but the mere accumulation of fluid, without pain or tenderness. The swelling may spontaneously disappear, but usually important and destructive alterations occur in the joint, the cartilages are destroyed, the ends of the bones worn off, and partial and entire luxation results.* The bones of the body of an ataxic manifest an extreme fragility and break easily.

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* "Diseases of the Nervous System," p. 138.

Course, Duration, and Termination.—Beginning obscurely and developing slowly, it may be years before the character of the symptoms will justify the attempt at a diagnosis. The first stage lasts from several months to several years. The ataxic disorders usually begin in the lower extremities, and the pains are most severe in the part or member destined to become ataxic. It occasionally happens that the incoördination begins in the upper extremities. The second stage is even more protracted than the first, and its duration is an affair of years. When extension takes place to the upper extremity, the progress is usually more rapid. The whole duration of the disease is on the average seven years (Topinard), but many continue thirty years. The shortest duration of a well-observed and carefully recorded case is three years. The progress is affected by the seasons, the atmospherical conditions, and by the regimen. Sometimes ameliorations occur without any apparent cause, or the disease remains absolutely stationary for long periods ; then exacerbations are experienced. The final result may be determined by acute congestion or softening of the cord, by cerebral diseases, by extension to the anterior cornua and the evolution of progressive muscular atrophy, by gastro-intestinal inflammation, by cystitis and pylonephritis, by bed-sores, and by various intercurrent diseases. The most frequent of the intercurrent maladies is phthisis, for we find that, in a collection of forty-three cases, thirteen were terminated by consumption, four by broncho-pulmonary inflammations, two by enteritis, three by typhoid fever, etc. That a cure of a genuine case, extended to the second stage, is ever affected, seems very doubtful. That the disease may be arrested, after more or less damage has been inflicted, is perfectly true. The author has mentioned a case in which all the symptoms of the second stage were present, and which recovered completely under iodide of potassium, but the patient was a gilder.

Diagnosis.—The recognition of this disease is easy when fully developed. During the first stage, the pains may not be different from those of rheumatism or myalgia, but the occurrence of double vision and of sexual disorders should suggest their real character. At this period the sexual disorders are confounded with "seminal weakness," but the diagnosis ought to be made, by the pains, the double vision, and the time of life at which the nocturnal losses began. From all acute affections of the spinal cord this disease is separated by the exceeding slowness of its development as well as by the character of the attendant phenomena. From chronic myelitis and all other affections of the cord, accompanied by paraplegia, with or without wasting, locomotor ataxia is differentiated by the condition of ataxia. In the one, the muscles are paralyzed ; in the other, they are not paralyzed, but incoördinate. These coarse phenomena seem sufficient without entering into the numerous finer points of difference.

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* "Diseases of the Nervous System," by J. M. Charcot, Syd. Soc. ed., London, 1877, p. 97. See also "Spinal Arthropathies," by Weir Mitchell, "American Journal of the Medical Sciences," April, 1875.

Treatment.—If the disease is recognized early, before important changes have occurred in the spinal cord, the first of all remedies is *rest*, and as nearly absolute rest as possible. The results accomplished in this way are remarkable. The patient should avoid all use of his muscles, and should remain recumbent for weeks. The rest-cure involves the complete severance from all cares, occupations, and movements for a period of two or three months, and subsequently greatly modified occupation and movement for some months longer. The position should be on one side and toward the face as much as possible, and on a firm bed or lounge, without any constricting clothing. The diet must be light and simple, corresponding to the changed necessities of the organism. Coffee, tea, tobacco, and alcoholic stimulants should be given up. Next to rest in importance is the cold-water cure, which may be well conjoined with the rest-cure, and thus serve a double purpose. Erb says the "thermal baths" are hurtful, but that the results of the "cold-water baths are extraordinarily favorable. . . . Of nineteen tabes patients who went through with the cold-water cure, sixteen experienced more or less benefit, two saw no improvement, and only one grew slightly worse." The temperature of the water must not be below 68° Fahr., nor above 88° Fahr., and the application should consist of the rubbing wet pack confined to the spine, the wet cold compress applied along the spine for some minutes, and cold sponging of the spine, all cold douches and full baths being avoided. The treatment may be conducted better at home, if the patients are provided with the means. The springs of our mountain-regions of Virginia, Pennsylvania, New York, etc., may be advised during the summer and fall, the *temperature and not the composition of the water being heeded.* The author has seen a great deal of injury done by the hot springs of Arkansas in this disease. The third remedy is galvanism, direct continuous currents to the spine, labile applications to the extremities for the relief of pain, faradic currents to wasting muscles, and to the bladder if paralyzed. As regards the internal medicines, the use of iodide of potassium is proper in every case for a short time, lest there may be a syphilitic or metallic lesion of the cord. If no results follow in two or three weeks, a continuation of the remedy will not be advantageous. If there be a decided decline of the vital powers, the best results are obtained from lactophosphate of lime and cod-liver-oil. Nitrate of silver has been serviceable in many cases, and is placed first as a remedy by some great authorities, but the danger of staining the tissues of the body is very great. The author has had excellent results from the persistent use of the chloride of gold and sodium, and when there is reason to suspect a syphilitic taint, he has conjoined with it minute doses of corrosive sublimate. Arrest of the disease has been, apparently, obtained in some instances, no further developments occurring after one and two years of close observation. Phosphorus has produced good effects in the hands of Dujardin-Beaumetz, but has been less useful in the experience of others. Nerve-stretching has now been done in many cases, with much relief. The sciatics are exposed, and stretched by the finger or a hook placed under them. Unfortunately, the first encouraging experiences have not persisted, and the operation of nerve-stretching, as a remedy for tabes, is rapidly declining in professional estimation.

LATERAL SPINAL SCLEROSIS.

Definition.—This term is employed for uniformity to express a disease having similar lesions to those of posterior spinal sclerosis but a different seat. By Charcot this disease is named *spasmodic tabes dorsalis*, and by Erb *spastic spinal paralysis.*

Pathogeny.—Lateral spinal sclerosis develops under the same conditions as posterior spinal sclerosis. The site of the lesions is the lateral white columns, and the changes consist in the gray gelatiniform degeneration. There occurs an interstitial hyperplasia of the connective tissue, and an atrophy of the proper nerve-elements. Although it chiefly affects the posterior part of the lateral column, it may extend forward to the anterior horn (its external angle), posteriorly to the anterior gray matter, and internally to the deepest portion of the lateral columns (Charcot). Secondary degeneration of the lateral columns, which occurs in certain cerebral diseases, is found on one side only. In the disease described by Charcot under the name *amyotrophic lateral sclerosis*, to the sclerosis of the lateral columns are added atrophy and disappearance of the multipolar ganglion-cells of the anterior cornua. This form of spinal sclerosis is situated in the cervical enlargement by preference (Erb). Lateral spinal sclerosis has its seat in the whole length of the cord—from the lumbar region up to the medulla oblongata.

Symptoms.—The symptoms of this disease are peculiarly striking, in that paraplegia exists with motor irritation. Before the motor symptoms there may be present such sensory disturbances as pain in the back, tingling, formication, and "tearing pains," but these are usually transient. The irritation symptoms are motor, and consist of jerking and twitching, cramps, and stiffness of the muscles, felt especially after fatiguing exercise, and at night on lying down. The muscles gradually become very tense, and certain movements difficult in consequence. Because of the continuous tonic contractions of the muscles the knees seem stiff, the step is shortened, and the legs approximated. The gait is a hop, the patient stepping on the toes, and showing a tendency to fall forward. This peculiarity of muscular movement is due, not altogether to the tonic extension state of the muscles, but to paresis. At first there is a feeling of heaviness and weakness, the muscles becoming very tired on slight exertion, and this passes on into paresis, only in very rare cases into paralysis. When the point of the foot rests on the floor, the patient sitting, a tremor of the limb is produced. The tendon reflexes in this disease are much exaggerated. The sensibility is unaffected; there is no atrophy of the muscles; and the functions of the rectum, bladder, and sexual system remain unaltered. The disease, beginning below, extends gradually upward. When the muscles of the trunk become affected, sitting up, or rising from the recumbent to the upright posture becomes difficult, finally impossible. When the arms are involved, the same combined weakness and rigidity, increase of the reflexes, paresis and contractures occur. But there are no symptoms of ataxia, and paralysis only rarely results. Sometimes the disease assumes a hemiplegic form, passing from one lower extremity to the corresponding upper extremity. When the disease completes its development, so to speak, it remains nearly stationary for many years, yet in most cases, ultimately, the contractures increase, and the paralysis becomes complete, and the patient is then entirely disabled. Nevertheless the malady does not prove fatal of itself, the termination being by some intercurrent disease. In that form of anterior spinal sclerosis in which the lesions involve the anterior cornua, and which is accompanied by progressive muscular atrophy, the symptoms present are those of anterior spinal sclerosis and progressive muscular atrophy.[*] As the anatomical site of the disease is the cervical portion of the cord, the symptoms first produced are those of the upper extremities. The muscles of the arms are occupied by fibrillary contractions, are wasted, paretic, but still retain the electro-contractility. The muscles of the arms, jaws, and neck are also in a state of tonic contraction passing into contractures, which ultimately disappear when the changes in the muscles are complete. In from four months to a year both arms are fully affected, and then extension takes place to the lower extremities. The same phenomena of paresis and rigidity with wasting take place in the lower extremities, but the bladder and rectum are not affected. Then occur also in the lower limbs the fibrillary contractions and clonic spasms, with permanent muscular rigidity, which are characteristic of this disease. In the third stage, the field of morbid activity is transferred to the medulla oblongata. Respiratory and circulatory disturbances then ensue, and death speedily occurs. The whole course of this disease is completed in from two to three years.

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[*] "Deux cas d'atrophie musculaire progressive avec lésions de la substance grise et des faisceaux antérolatéraux de la moelle épinière," par MM. J. M. Charcot et A. Joffroy, "Archives de Physiologie," vol. ii, 1869, p. 354, *et. seq.*

Diagnosis.—The main points of difference between *posterior* and *anterolateral* spinal sclerosis have been referred to in passing. The presence of the reflexes, the absence of all symptoms of ataxia, weakness instead of incoördination, the contractures and clonic spasms—all characteristic of lateral, are wanting in the posterior sclerosis.

Treatment.—The principles and methods of treatment are the same as in posterior spinal sclerosis, which have been sufficiently set forth in the preceding chapter.

INFANTILE PARALYSIS—POLIOMYELITIS ANTERIOR ACUTA OF ADULTS.

Definition.—By *infantile paralysis* is meant a peculiar form of spinal paralysis, occurring in children suddenly, and due to an inflammation of the anterior horns of gray matter. It is now known that the same form of disease occurs in adults also, though much less often.

Causes.—Infantile paralysis, as the name implies, is a disease of early life, and occurs most frequently from six months to the fourth year; but precisely the same form of disease occasionally is encountered up to sixty years of age, so that the term proposed by Kussmaul —*poliomyelitis antèrior acuta*—is more appropriate. Besides age, little is known as to the causes producing this disease. The influence of summer heat seems established by the observations of Sinkler.* As cases frequently occur during the course of convalescence from the exanthemata, and other acute febrile affections, a causative relation is supposed to exist between them. The important negative fact, that the influence of heredity can not be traced, must be stated.

Pathological Anatomy.—The naked-eye appearances furnish no exact information, and may be entirely negative. On microscopic examination, important changes are found in the anterior horns of gray matter, in the dorso-lumbar and cervical enlargements of the cord. The change consists in an inflammatory softening; the nerve-elements are disassociated by an exudation containing numerous granulation corpuscles and free nuclei; the neuroglia undergoes hyperplasia, and the blood-vessels are abnormally distended; the multipolar ganglion-cells have wasted, and many disappeared, while those remaining are in various stages of atrophic degeneration. The softening occurs in certain areas, from a half-inch to an inch in length, and on both sides, or on one side only, and especially in the dorso-lumbar enlargement. The softening extends a little posteriorly and laterally, and sclerotic degeneration also occurs in the adjacent antero-lateral columns. Similar changes take place in the anterior roots. Extensive wasting, atrophic degeneration, and sclerosis, occur in all cases and after many years. The anterior nerve-roots are thin, atrophied, and translucent, and more or less degeneration takes place in the filaments of the peripheral nerves. The muscles to which the nerves are distributed undergo very serious alterations, which consist in an increase of the connective tissue, the formation of numerous fat cells and granules, and the degeneration and disappearance of the muscular fibers. The bones of the paralyzed members cease to grow, and degenerate more or less, the cancellated structure being relatively increased, and the fatty tissue also. Important changes occur in the joints ; the articular surfaces are atrophied and eroded, the ligaments thinned and stretched, the articulations relaxed. By reason of these atrophic changes great deformities, the worst forms of club-foot, are produced.

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* "American Journal of the Medical Sciences," vol. lxix, p. 348.

Symptoms.—The usual onset of this disease is a fever, which lasts a day or two, and on recovery from which it is observed, with surprise, that the child is paralyzed. The fever may be accompanied with headache, pain in the back and limbs, with vertigo and delirium, in some cases with convulsions. Dr. Mary Putnam-Jacobi * has analyzed one hundred and sixty-three cases, and finds that there are several modes of onset. In twelve of these cases the paralysis occurred suddenly without any prodromes ; in some cases the paralysis appears in the morning after a quiet night, or between morning and evening, without symptoms ; in the majority of cases there is an attack of fever lasting two or three days ; in some, merely nausea and vomiting, and in still others the paralysis is preceded by convulsions. What symptoms soever may precede the palsy, they subside in a day, or in two or three days, and the health seems restored, but one limb or several are found to be paralyzed ; or one leg is limp and motionless, and in an hour or two the other leg is found to be in the same condition ; and, in the course of the next twenty-four hours, the arms may also be paralyzed. From the beginning of the symptoms until the paralysis is completed, rarely more than a week is required. The bladder may participate in the paralysis, and the urine be retained, or there may be incontinence, but the bladder is not permanently affected, and these troubles disappear in a few days or weeks. Sensibility is not affected. The paralysis is complete at once, and soon begins to lessen, some restoration of power taking place in from one to three weeks, which may gradually go on until the paralyzed parts are completely restored in the course of a few months. During this period the electro-contractility and the nutrition of the muscles are not affected in this group of cases, although the muscles are flabby and soft. Most of the cases behave differently. Improvement begins as in the cases just narrated, but it proceeds to a certain point only; some of the members recover entirely, leaving one or more or a single group of muscles affected. Thus the arms may be restored and the lower limbs continue paralyzed, or one arm or one leg may remain disabled. Rarely is one half of the body (hemiplegia) affected, and, if such be the case, the cause is to be sought within the cranium. When an arm is alone affected, the extensors of the arm and fingers are paralyzed; when the lower limbs are involved, the disability is in the extensors of the thigh (the psoas, Rosenthal), or in the muscles supplied by the peroneal nerve. The muscles remaining paralyzed are affected permanently, and by a rapidly progressive atrophy; the tendon and other reflexes and the electro-contractility to the faradic current are abolished (reactions of degeneration). The temperature of the paralyzed parts falls several degrees; they become cool to the touch, and present a blue, cyanosed appearance. The muscles waste till there is nothing but connective tissue and fat, the joints change in form and structure, the growth of the limb is arrested, and, if one of the lower limbs is affected, assuming often one of the forms of club-foot. Seguin* has given a careful analysis of many of the cases of spinal paralysis (poliomyelitis anterior acuta), which have been published. The following symptoms he regards as characteristic: "Dysæsthesia, and slight temporary anæsthesia, paresis and akinesis, both these symptoms affecting the extremities, and in rare cases the eyes, face, tongue, and throat; not affecting the respiratory muscles, nor those of the back and abdomen, nor the bladder, nor the sphincter ani. Muscular atrophy in the paralyzed parts. Loss of electro-muscular contractility (to faradic current) in the atrophied muscles. A strong tendency to spontaneous retrocession of the palsy, and to spontaneous cure. The important negative characters of this affection are: absence of palsy of the bladder, or of the sphincter ani, or of the respiratory muscles; no bed-sores; no great and extensive anæsthesia; no spinal epilepsy."

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* "The American Journal of Obstetrics," June, 1874.

Acute Poliomyelitis of the Adult.—Although essentially the same disease in the adult as in children, it presents in the former some special characteristics, owing to the difference in bodily development. It begins with more or less intense headache, backache, nausea and vomiting, hebetude of mind, and even mild delirium. Various disorders of sensibility are noted by the adult when they can not be got from children. These disorders of sensation are numbness, formication, tingling, in the parts subsequently paralyzed. There is usually considerable fever, the temperature rising to 102°–103° Fahr., but this symptom may be wanting. The paresis or paralysis comes on in a few hours after the beginning of the symptoms, and is widespread as a rule, but may be confined to a few muscular groups. The affected muscles are flaccid, and waste; the reflexes are much less active or disappear entirely; the reactions to the faradic current are feeble or are entirely abolished, the reactions of degeneration appearing. In the adult, however, more frequently than in children, the muscles retain their power of response to faradic stimulation, although in a feeble degree.

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* "Spinal Paralysis of the Adult," New York, 1874, p. 27.

When the systemic disturbance subsides, in a few days, the paresis and paralysis manifest a tendency to restoration, which may, indeed, be complete after some weeks or months, but frequently some of the muscles undergo atrophic degeneration, with the characteristic electrical reactions, and deformities result, but never to the same extent as in children. In a majority of the cases, the resulting paralysis involves all of the members, or both of the upper, or both of the lower extremities. Paresis of the sensory nerves may be present at first, but this soon disappears, and thereafter sensibility continues normal. The special senses remain unaffected. Some weakness of the bladder is noted at the outset, but this is a temporary symptom. No change takes place in the sexual functions.

Course, Duration, and Termination.—The course of the disease is very uniform. The mildest cases, in which restoration of power begins in a few days, recover entirely in a few weeks or in a month or two. These cases have been designated "temporary paralysis." Other cases, in which a single member or a group of muscles remains paralyzed after the efforts at restoration have ceased, may regain the lost power in from two to six months. If the restoration does not take place within this time, it becomes less and less likely with the increasing duration of the case. Partial restoration is the rule even in unfavorable cases. Much depends on the treatment. So far as danger to life is concerned, the prognosis is always favorable. So far as ultimate entire restoration is concerned, the prognosis is doubtful. Persistent and rightly conducted electrical treatment may accomplish much even in the worst cases.

Diagnosis.—The first point in diagnosis is the condition of the paralyzed muscles. If wasted, how far do the muscular elements exist? This is ascertained by electrical tests. In these cases the muscles do not respond to a faradic current, but will contract on the application of a weak and slowly interrupted galvanic. Muscular contraction is the proof of the presence of the muscular elements. By the use of the harpoon, some portion of the tissue may be withdrawn and submitted to a microscopic examination. Infantile paralysis may be confounded with *acute myelitis, hæmorrhage into the cord, progressive muscular atrophy, paralysis from cerebral affections* in childhood and *paralysis* *from local nerve-lesions.* From myelitis the distinction is made by the stage of excitation affecting sensibility and motility, and the stage of depression also affecting sensibility and motility and the bladder. From hæmorrhage, the distinction is made first on account of its rarity, next the suddenness of the attack, sensibility being destroyed as well as motility, usually, and the sphincters paralyzed. From progressive muscular atrophy, the distinction is made by the age of the subject, the slow development, and the affection of isolated muscular groups in turn. From cerebral lesions, the distinction is made by the pronounced cerebral symptoms, by the hemiplegia, by the electrical reaction, the electro-contractility rather heightened than lost, and by the appearance and condition of the paralyzed members. From paralyses due to local injury of nerve, the distinction is made by the history of the case, the evidence of injury, by the absence of fever, by the diffusion of the paralysis at first, followed by localization.

Treatment.—During the attack of fever with which the disease begins, only symptomatic treatment is proper, since a diagnosis is not possible. When paralysis has occurred the damage to the cord is complete, but, as the functional disturbance is more extensive than the symptomatic expression of the real lesions, the improvement which follows from the first paralysis is simply the disappearance of the merely functional troubles. Any active treatment, therefore, instituted with a view of combating an inflammation, is improperly applied. The problem is to prevent further destruction of the gray matter, and to restore damaged but still functionally capable tissue. The remedies best adapted to accomplish this, and which in the author's hands have acted best, are quinine and belladonna (from a fourth to four grains, according to age, of quinine, and from $\frac{1}{20}$ to $\frac{1}{4}$ grain of belladonna extract); hot douche to the spine and tepid wet packs; the application of galvanism, inverse current, stabile, large volume and low intensity, and rest, as nearly absolute as possible, until the period of restoration. When the period of improvement comes on, the muscles must be faradized, if they react to the faradic current, or galvanized if they react only to the galvanic current. Massage is suitably combined with electrical treatment. The wasted muscles are much improved by aquapuncture; still more by the intramuscular injection of strychnine ($\frac{1}{100}$—$\frac{1}{60}$ grain) two or three times a week. The injections of strychnine should not be practiced until after the period of restoration—the stationary period.

PROGRESSIVE MUSCULAR ATROPHY.

Definition.—By the term *progressive muscular atrophy* is meant a gradual and progressive wasting of the voluntary muscular system which pursues a certain defined course.

Causes.—Numerous examples of hereditary transmission, some of them very remarkable, have been reported. The male sex is much more susceptible, and this is equally the case when the disease is hereditary. The most active period of life—from thirty to fifty—is the period of greatest liability; but youth and early manhood are by no means exempt, cases occurring before ten. Powerful muscular exertion, or overstrain of a group of muscles in certain occupations, seems to excite the disease; and in children the disease is invited to the lower limbs by prolonged effort on the legs. Exhausting diseases, the poisons of lead and syphilis, and certain dyscrasiæ, seem to exert an influence in developing the disease. Exposure to cold and mechanical injuries have apparently given rise to progressive atrophy.

Pathological Anatomy.—The morbid alterations are of two groups —spinal and muscular. The changes in the spinal cord are similar to those which take place in the spinal arthropathies in general, i. e., atrophy and degeneration of the anterior columns, wasting and disappearance of the multipolar ganglion-cells, of the anterior horns, hyperplasia of the neuroglia, corpora amylacea, granule-cells and fat-corpuscles. The anterior roots are similarly affected—are wasted, atrophied, and degenerated. In one third of the reported cases in which the cord was examined, no changes were found of any kind. The alterations in the muscles have been most elaborately studied by Friedreich,* who holds to the muscular origin of the disease. He asserts that the initial change consists in an inflammation with hyperplasia of the interstitial connective tissue uniting the primitive bundles. Morbid changes occur in the primitive bundles: proliferation of the nuclei and multiplication of the muscular corpuscles. Wasting of the muscular substance goes on, *pari passu*, with the increase of the connective tissue, and fatty degeneration contributes to it. The final result is, that the muscle is converted into a mere fibrous band with numerous fat-cells, the development of this latter material taking place outside of the muscular elements and in the newly formed connective tissue. The theory of Friedreich, which he maintains with remarkable skill and learning, is that the disease begins in the muscles, the intramuscular nerves are next affected, and an ascending neuritis conveys the morbid process to the spinal cord, which becomes in turn diseased. The other view is, that the changes in the muscles are secondary to the morbid process in the spinal cord, especially in the multipolar ganglion-cells of the cornua.†

Symptoms.—According to Friedreich's statistics, of one hundred and forty-six cases, there were one hundred and eleven instances of the disease beginning in the right upper extremity, twenty-seven in the lower, and eight in the lumbar muscles. Sometimes the tongue, sometimes the palate muscles, an example of which the author has seen, are first affected. The first dorsal interosseus is usually the first muscle attacked in the upper extremity, then the muscles of the thenar and hypothenar eminence, the deltoid, etc. Sometimes the pectoralis major and serratus magnus are the first to undergo atrophy. In children the lumbar muscles are usually the first to atrophy, the degeneration taking the form of pseudo-hypertrophic. The loss of volume which the muscles undergo is not always a measure of the real degeneration, since a very considerable hyperplasia of the fatty tissue sometimes takes place, with the effect to increase the apparent size. The next symptom is fibrillary contraction: the muscle undergoing atrophy so long as it remains, is agitated by fine tremors, which consist in waves or oscillations of movement of the muscular fibrillæ. If, now, the muscles of the diseased hand are tested by the dynamometer, they will be found extremely weak as compared with the sound hand. The hand also becomes greatly deformed, rigid, and claw-like, presenting the appearance of a bird's talons. The electro-contractility is preserved so long as muscular fibers remain to be stimulated, but the reaction to the galvanic persists for some time after the faradic excitability has disappeared. In most patients a good deal of pain is experienced in the muscles about to be affected and during the process of wasting, but the sensibility to pain and to temperature diminishes to below normal in the last stages. The temperature of the wasted parts is also reduced several degrees, and they are cold to the touch; and the integument appears normal or pale, or blue, and cyanosed. The perspiration is usually increased in the affected member or part, and sometimes generally. Changes in the joints, comparable to those which take place in locomotor ataxia and other spinal diseases, are also observed in progressive muscular atrophy.* Changes in the pupil and other oculomotor phenomena occur when progressive muscular atrophy is associated with glosso-labio-pharyngeal paralysis. This disease may be accompanied with fever during the first weeks or months, often associated with the joint-lesions. How far this is accidental or a necessary part of progressive muscular atrophy does not appear to be well understood.

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* "Ueber progressive Muskelatrophie," etc., von Dr. N. Friedreich, Berlin, 1873, cap. ii, p. 46.

† Charcot and Joffroy, "Archives de Physiologie," vols. ii and iii, *op. cit.* A. Hayem, ibid. See also, as explanatory of spinal affections consecutive to nerve-injuries, A. Vulpian, ibid., p. 221.

Course, Duration, and Termination.—The course of this disease is extremely protracted in many cases. The manner of spread of the myopathic process is not in accordance with a uniform plan. It sometimes extends by contiguity of tissue, sometimes leaps over groups of muscles to attack distant muscles. The extension is limited by the larger joints. Beginning in the hand, an extension to the arm does not take place; some of the extensors of the forearm undergoing atrophy, the muscles of the arm are not attacked; the deltoid and arm muscles affected, the elbow-joint is not passed; similarly in atrophy of the leg-muscles, the knee-joint seems to prevent extension to the thigh. Some muscles are never affected; those of the head are not often; and, when the tongue and lip muscles and the laryngeal muscles are affected, the disease is complicated with glosso-labio-laryngeal paralysis. The diaphragm and the respiratory muscles and the accessory muscles of respiration are finally invaded. Death then ensues by hypostatic congestion and œdema of the lungs. When the larynx is invaded, the voice is lost, and there is difficulty of breathing from cessation of the laryngeal movements. The muscles of the ear may also be invaded, and impaired hearing result. Friedreich gives a remarkable example, pictorially represented, of a man all of whose voluntary muscles are wasted, and who seems to retain alone the power of breathing. The march to this end is exceedingly slow, unless, as is not unfrequently the case, the morbid process involves the anterior cornua of the medulla oblongata, the effects of which have already been described. At first no trouble is produced by the wasting of the muscles of the extremities; the general health does not suffer; the powers of body and mind are otherwise adequate to their work. Sometimes the disease is arrested, and remains stationary for years. A few cases are terminated by bed-sores; many by intercurrent maladies, of which pulmonary tuberculosis is the chief.

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* On this point consult Weir Mitchell's "Spinal Arthropathies," in "American Journal of the Medical Sciences," April, 1875, p. 339.

Diagnosis.—A fully formed case can never present any difficulty in this respect, but at the initial period there may be doubt whether the wasting is due to local injury, injury of the nerve-trunk, or the result of rheumatism. The distinction rests on the pains, the fibrillary trembling, and the absence of any local cause to account for the atrophy.

Treatment.—Nothing has ever been accomplished by the use of internal medicines. The author has apparently effected great improvement in a case, confined as yet to the left upper extremity, by the injection of glycerine solution into the wasting muscles. The strength of the solution is one third glycerine, and it is injected three times a week. The two remedies of unquestionable utility are galvanism and massage. The author has had good results from galvanism, and he can not share the despondency of authors generally in regard to its utility. Erb reports favorably as to the good effects of the galvanic current. Strong currents must be used to excite vigorous contractions for a brief period—two minutes. A descending current should also be applied to the whole length of the spine, daily, for a minute or two. Massage, using with friction a fat, preferably lard, is also highly serviceable. This should consist of friction, kneading, and tapping the muscles. Hot douches to the spine and the rubbing wet pack for the affected members are also to be highly commended.

PSEUDO-HYPERTROPHIC PROGRESSIVE MUSCULAR ATROPHY.

Pathogeny and Symptoms.—This disease differs from progressive muscular atrophy, in the remarkable fact that the atrophied muscles increase in size, and are apparently hypertrophied, because of an hyperplasia of the connective and fatty tissue. The anatomical change consists, in brief, in a proliferation of the connective tissue between the fibrilla (Friedreich) and the adventitia of the small vessels. The newly formed connective tissue is remarkable for the number of its cells and nuclei, which are transformed into fat-cells. As the connective tissue develops the muscular elements disappear, or at least only in part remain, much altered, and thinner. Now and then are encountered some muscular fibers which have undergone hypertrophy. The muscular elements are also invaded by an irritative process, become granular and degenerate, so that the atrophy is not wholly a simple atrophy from overgrowth of the connective tissue. When the process is complete the muscles present a grayish or yellowish-white appearance, and can hardly be distinguished from the adjacent fatty and connective tissue.

This disease occurs almost wholly in childhood, and before ten years of age. In eighty cases, it began from the first to the fifth year in forty-five; from the sixth to the tenth, twenty-two times; from the eleventh to the sixteenth, eight times; and in five cases it occurred from the twenty-second to the forty-third year (Erb). Hereditary influence plays a very important part in the development of the disease. Other causes have been assigned, and probably with little reason, for all the facts go to prove the existence of a peculiar neurodiathesis.

The morbid process begins in the lower limbs—chiefly in the legs, although it may begin in the thighs. Before the hypertrophic enlargement manifests itself, muscular weakness has occurred; fatigue is quickly experienced; the legs trip easily and give way; the gait is awkward. After a time a child thus affected is not able to rise, when down, unless aided, and can not walk unless steadied; the gait assumes a straddling manner, somewhat like that of a duck, and when the thigh-muscles are affected he can not rise unless he supports his thighs by his hands, and in sitting down can not control the act, but plumps down suddenly. When recumbent, the legs are wide apart, the soles of the feet turned toward each other, the heels drawn up, and the knee and hip joints flexed. All the movements of the foot are imperfectly executed, except flexing the toes; the movements of the thigh are equally imperfect, except mere flexion of the knee. The position in standing is very characteristic: the lumbar portion of the spine is greatly incurved (lordosis), the dorsal portion bent outward (gibbosity). The diminution in power offers a remarkable contrast to the enormous bulk of the affected members. If the disease attacks the upper extremity, it takes the form of progressive muscular atrophy, and the two may exist together. Before the muscular tissue has disappeared, the same fibrillary twitchings occur as in the other form of the disease. The electro-contractility declines progressively with the diminution of the muscular elements, and in this disease the more decidedly because of the great collection of fatty and fibroid tissue overlying the muscular elements. There is more or less pain experienced by these patients, in the back, and through the parts to become affected. The temperature declines several degrees in the hypertrophied and atrophied parts. The termination of these cases has been by some intercurrent disease, usually of the respiratory organs.

CHRONIC POLIOMYELITIS ANTERIOR—ATROPHIC SPINAL PARALYSIS.

Definition.—As there is an acute poliomyelitis affecting the anterior columns, so there is a chronic disease of similar character, but possessing some distinctive features. It consists in a chronic atrophic degeneration—a muscular paralysis with wasting and atrophy of the affected muscles—secondary to degeneration of the gray matter of the anterior horns.

Causes.—While the acute anterior poliomyelitis attacks children more especially, although occasionally appearing in adults, the chronic affection occurs at the middle period of life—from thirty to fifty years of age. As very similar symptoms and lesions are caused by lead, copper, and some other metals, the modern use of these metals in domestic life may have a causative relation to this and corresponding nervous affections. Injury to the spine, excessive fatigue, exposure to cold and damp combined, sexual and alcoholic excess, have all been supposed to bring on this disease. In any case thus produced it is quite certain that a neuropathic type of constitution must have existed.

Pathogeny and Symptoms.—The seat of the pathological changes and their general character correspond to those of acute anterior poliomyelitis. The multipolar ganglion cells are degenerated and wasted, the vessels are thickened, the perivascular lymph-spaces crowded with leucocytes, red corpuscles, and granular matter, and the anterior nerve-roots more or less advanced in atrophy. The paralyzed muscles exhibit the characteristic degenerative changes, consisting in atrophy and disappearance of the muscular elements, and the substitution of connective and fatty tissue.

The symptoms develop rather insidiously, with a sense of fatigue and exhaustion of the lower limbs, pain in the back, loins, and hips, headache and some slight feverishness, with the anorexia and general *malaise* belonging thereto. Then some tingling, creeping, and crawling sensations, "pins and needles," and distinct muscular weakness are experienced. The weakness gradually deepens into a paralysis, a group of muscles or an extremity loses its power entirely, and next other muscular groups, or a member, become paralyzed. As a rule the morbid process begins in a lower extremity and extends to the upper members, but the process may be reversed, and the paralysis beginning above may extend downward. The flexors of the foot first, the flexors of the leg and thigh next, and then the extensor of the limb are affected. In the upper extremity there is no regular order for the paralysis to occur: the muscles of the hand—the flexors of the fingers and hand; again, the extensors of the forearm, are first paralyzed, and the process extends thence to the arm and shoulder-muscles, until all become paralyzed and wasting. Ultimately, the muscles of the back and of the abdomen, and sometimes, also, of the chest, are paralyzed, so that the breathing becomes difficult because carried on by the diaphragm, and the expulsion of the fæces and urine is inefficiently performed. When the paralysis reaches this extent, the patient is quite helpless, and can not maintain the sitting posture. The functions of organic life are, however, carried on in the normal manner. Digestion, assimilation, the circulation, the sexual functions, the bladder and rectum, remain unaffected. Sensibility is not impaired, and bed-sores do not form. The reflexes decline and disappear with the progress of the changes in the spinal cord and muscles. The electric reactions are changed from the normal in harmony with the anatomical alterations. As the muscles atrophy, faradic excitability declines quantitatively, and presently ceases. The galvanic excitability increases in accordance with the formulæ of the reactions of degeneration. But galvanic excitability disappears finally in those muscles so far atrophied that none of the proper anatomical elements remain.

Course, Duration, and Termination.—The progress of this affection varies in rapidity between wide limits. In some cases paralysis supervenes rather suddenly soon after the initial symptoms appear; in others, after the first weakness and heaviness of the lower limbs, months, even years, intervene before complete paralysis occurs below and extends to the upper limbs. In the cases which take a favorable direction, the upward extension of the morbid process is arrested. If the changes in the cord continue to advance, after a time the medulla oblongata is reached, and then there is experienced that group of symptoms characteristic of disease of this part—as ataxia of speech, difficulty of swallowing, embarrassed respiration, cardiac failure, etc., and death from asphyxia. A considerable proportion of the cases improve: the upward extension of the disease is arrested; the paralysis slowly lessens and voluntary power gained, the electrical reactions changing accordingly—first, the abnormal galvanic excitability gradually declining, while the faradic excitability is recovered. It is by almost imperceptible gradations that improvement takes place, and although complete recovery does occur, more frequently there remain behind disabilities and deformities, the result of permanent changes in certain nerves and muscles, especially of the leg and foot.

Diagnosis.—The disease with which chronic anterior poliomyelitis is most apt to be confounded is *progressive muscular atrophy*, but the distinction between them is made by reference to the mode of onset, progress, and termination. The latter begins silently without systemic disturbance. The paralysis follows the atrophy in the latter, while in the former precedes it. The electrical condition of the wasted muscles is different ; in progressive muscular atrophy the muscles react until far advanced in degeneration, but in chronic anterior poliomyelitis the reactions of degeneration appear soon after the paralysis occurs. In respect to rate of progress, duration, and mortality, progressive muscular atrophy differs widely from the other, in that it is slower in progress, much longer continued, and is fatal, whereas chronic poliomyelitis anterior frequently gets well.

Treatment.—The reader is referred to the section on acute anterior poliomyelitis for the method of treatment, which is as applicable to this disease.

ACUTE ASCENDING PARALYSIS—LANDRY'S DISEASE.

Definition.—This disease, as its name implies, is an acute motor paralysis, usually ascending, and involving all parts of the voluntary muscular system, with wasting of the affected muscles, but without change in the electrical reactions. It is entitled "Landry's Disease," because he first accurately described and differentiated it, although it had been observed before.

Pathogeny and Symptoms.—After exposure to cold, or in consequence of alcoholic excess, or because of the presence of metals in the system, or excited by some unknown cause, the patient, who is usually between twenty and forty years of age, is seized with a little fever, pains in the back and limbs, numbness, tingling, and a sense of extreme fatigue. These symptoms continue a few days—rarely several weeks ; next come on extreme weakness of the feet, then of the legs, of the thighs, until in a few days there is complete paralysis of the lower extremities ; the muscles are completely relaxed, and the legs lie inert wherever placed. It is very distinctly an acute ascending paralysis—for no sooner is the loss of power in the inferior extremities completed than the muscles of the trunk are invaded, and, even before the abdominal, the upper extremities become paralyzed, and, like the lower, lie relaxed and motionless—first, the hand in its complicated motions, then the forearm, and ultimately the arm and shoulder.

Not all cases are paralyzed in the order above described, and none are affected in strictly anatomical order—for in some few instances the paralysis begins in the hand and thence descends to the lower limbs, and in all cases the hands and feet are implicated before the parts most nearly connected with the spinal cord. Rarely a case occurs in which the medulla is first attacked, with the characteristic respiratory and circulatory disturbances resulting. According to Landry, the order in which the paralysis extends in the muscular system is the following: the toes and feet with the muscles acting on them; then the thigh and pelvic muscles, posteriorly, and, after these, the muscles in the front part of the thigh. A similar order is pursued in the upper extremity: first are affected the muscles acting on the fingers and hand; next those which move the arm on the scapula, and then those moving the forearm on the arm. Entirely after the muscles of the extremities, the trunk muscles are paralyzed; then come the muscles engaged in the respiratory movements, and after these, and in the order named, the muscles of the tongue, pharynx, and œsophagus. Attention to the mode in which the paralytic phenomena are developed will facilitate the diagnosis.

Sensibility is somewhat affected in many cases. There are numbness, tingling, formication, and impaired tactile and pain-sense, usually in the inferior extremities, but elsewhere the æsthesiometer discloses no changes. The reflexes are, as a rule, finally abolished, although at first there may be no change. The "knee-jerk" ceases. The bladder and rectum are not affected.

Whether the medulla oblongata be early or late affected, in every fatal case it is invaded by the morbid process, and its injury is the cause of death by asphyxia. The disease is of short duration for the most part, death occurring in a few days, or in a few weeks certainly, the average being ten days. It is not always fatal. The progress of the disease in favorable cases is arrested before the medulla oblongata is reached, although some cases presenting evidences of the functional derangement of this organ have, nevertheless, terminated in recovery. When improvement does take place, the change is early manifested by a cessation of morbid activity, by a return of function to the parts last paralyzed—the hands coming into use, then the arms, muscles of the chest, and abdomen, and finally of the lower extremities.

The rapid progress made by this affection, the preservation of the electric excitability of the muscles and of the functions of the rectum and bladder, the absence of bed-sores, and the prompt extension from below to the medulla oblongata, separate acute ascending paralysis from other acute affections of the cord, and render its diagnosis comparatively easy after the full development of the symptoms.

The prognosis, although grave, is not necessarily fatal. About one half of the cases, apparently, get well.

Treatment.—As some kind of toxic agency is supposed to underlie the morbid process, remedies should be addressed to the elimination of the poison. Syphilis, the minerals, will require large doses of the iodides. The rheumatic diathesis will need the remedies appropriate to that state. When the disease succeeds to some infection, as variola or typhoid, only the general condition can be taken into account. Galvanization of the cord has been apparently of benefit in a few instances. It is probable that the subcutaneous injection of strychnine would do good. It should be tried cautiously, and, if it act favorably, should be pushed, for, in the absence of specific lesions, a loss of power to functionate seems to be the essential condition of the spinal cord.

SOME DISEASES AFFECTING THE BRAIN AND SPINAL CORD.

MULTIPLE SCLEROSIS OF THE BRAIN AND CORD.

Definition.—By the term *multiple sclerosis of the brain and cord* is meant a disease characterized by the formation of isolated patches or nodules of sclerotic tissue in the brain, pons, medulla, cerebellum, and spinal cord. It is sometimes treated of as *cerebral sclerosis* and *spinal sclerosis*, but it becomes more and more apparent that neither organ is separately affected. By Charcot* it is entitled "disseminated sclerosis."

Causes.—In this disease both sexes are about equally affected, and it occurs from youth to middle age, becoming very rare after forty-five and before ten. The most powerful predisposing cause is heredity. Exposure to cold and fatigue, living in damp habitations, and sudden exposure of the body to cold and dampness when in a warm and perspiring state, are alleged to be causes, but doubts may well exist as to their influence unless a predisposition exist. Powerful and prolonged moral emotion, chagrin, anxiety, and other depressing moral causes, may favor the development of this affection. It occurs in the convalescence from acute infectious diseases.

Pathological Anatomy.—The disease in the brain and cord, to the naked eye, appear as glistening nodules underneath the pia. They are distinctly circumscribed, grayish patches, raised a little above the level of the cord sometimes, or depressed below, or on a level with the general surface, but always perfectly defined from the adjacent tissue. The patches are somewhat gelatinous and translucent, and marked by fine white lines, round or elliptical or irregular in shape, somewhat closely arranged, often confluent; dense, tough, almost cartilaginous in hardness; on section, rather glistening. The nodules vary greatly in size, from minute, microscopic objects up to the size of a walnut; in the brain they enlarge laterally; in the cord, in its long diameter. They vary greatly in number as in size, and are distributed widely through the brain and cord. In the brain they are found not in the gray but the white matter—in the white matter of the hemispheres, ventricles, optic thalamus, corpus striatum, peduncles, pons, cerebellum; in the cord, the nodules are found both in the gray and white matter and in the columns. The deposits occur in the nerve-roots and the nerve-trunks just as in the nerve-centers. The nodules themselves are composed of the neuroglia, much hypertrophied, a newly formed fibrillated connective tissue, remains of the nerve-elements, fat- and granule-cells, and corpora amylacea. In the nerve-fibers, the medullary sheath is first encroached on by the hyperplasia of the neuroglia, disappears by absorption, leaving the axis-cylinder, which in turn undergoes the sclerotic change, then disappears, so that ultimately nothing remains but the newly formed fibrous tissue containing numbers of so-called "spider-cells," free nuclei, corpora amylacea, and fat. Similar changes occur in the walls of the vessels, beginning in the adventitia and in the perivascular lymph-spaces. Ultimately the adventitia is closely united to the surrounding connective tissue, the other tunics are invaded by the hypertrophied connective tissue, nuclei form in great numbers, fatty degeneration occurs, the fat-elements crowding the perivascular lymph-spaces, and encroaching on the lumen of the vessels.

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* "Diseases of the Nervous System," "Sydenham Society Translation," lecture vi, p. 157.

Symptoms.—There are three forms usually described: the cerebral, the spinal, and the cerebro-spinal. But the description of this disease was purposely postponed to this point, as the spinal and cerebral forms rarely, if ever, exist separately, but the disease is cerebro-spinal sclerosis, in which, it is true, there may be a predominance of the cerebral or of the spinal symptoms in different cases, but in all the traces of both are discernible.

There are two modes of onset—a gradual and insidious mode, and a sudden and severe mode. When it begins slowly the symptoms may be chiefly cerebral or chiefly spinal: in the former, headache, vertigo, convulsions, or an attack of an apoplectiform variety, disordered and staggering gait, tremors in certain limbs or groups of muscles, impairment of special senses—of sight, of taste, of hearing, double vision, etc.; imperfect speech, and mental disorders of various kinds; in the latter (spinal form) there will be weakness and uncertainty of gait, ataxic disorders, numbness, tingling and pains in the extremities, incoördinate movements in writing, trembling, and severe attacks of gastralgia. This disease, as Charcot happily said, "is, in fact, an eminently polymorphic affection."* In the sphere of the sensory nervous system there are pains of various kinds, according to the position of the sclerotic nodules; pains in the face in the distribution of the fifth nerve, in the arms, and in the lower limbs, of an acute, lancinating character, with more diffused pains with a sense of pressure, constricting or girdle pain around the abdomen at different heights, with pains in the back and hips. Instead of pain, there is at a more advanced stage loss of sensation in various parts, or anæsthesia and analgesia. The sense of the position of members and of weight and resistance is also disordered or lost. There may be an entire absence of these sensations, and the appreciation of touch and pain continue normal. The disturbances in the motor sphere are more constant; first, motor weakness or paresis, which attacks one leg, then the other, and after a time the arms, or the order may be reversed; difficulty of locomotion, due not only to paralysis but to tonic contraction—the contraction of extension—which imparts to the gait a shuffling, dog-trot, or titubating character. The tonic contraction of extension passes into permanent contractures and rigidity. In many cases in which sclerosed nodules are largely deposited in the posterior columns the gait is incoördinate, and the usual phenomena of ataxia (reeling with the eyes closed, the peculiar gait) are present. Similar changes occur in the upper extremity, but the contractures and paralyses are usually hardly so pronounced as in the lower extremities. A very characteristic symptom is tremor, a *shaking tremor*, which occurs only during voluntary movement, and ceases when the parts are at rest. In the words of M. Charcot, "*the tremor manifests itself on the occasion of intentional movements of some extent; it ceases to exist when the muscles are abandoned to complete repose.*" Exceptional cases are encountered in which tremor is not present. It may have been present and then disappeared; it ceases when permanent contractions occur, so that the case can not be regarded as exceptional if the tremor is found on inquiry to have been present at some previous time and is now absent. The more powerfully the will is directed to the act, the more considerable and extensive the trembling. In conveying a glass of water to the mouth, the water is spilled and the glass rattles against the teeth. In any muscular act to which the attention is strongly attracted, not only the member acting, but the head, neck, and body are thrown into violent trembling. The reflexes are variously affected, and may be diminished or absent, but are often greatly increased, especially the tendon reflexes. Vesical, sexual, and rectal

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* *Supra*, p. 183.

disturbances only appear toward the end, when incontinence, impotence, and constipation will come on. While these symptoms from the spinal lesions are developing, characteristic cerebral phenomena also are occurring. The psychical functions are disordered. At first, changes of disposition are noticed, the emotional centers becoming easily excited, and laughing and weeping occurring with equal readiness; irritability of temper and unexpected gusts of anger are common. Memory is early impaired, and reason, judgment, and the power to acquire knowledge are much weakened. Presently distinct forms of mental derangement make their appearance, as melancholia, mania with exaltation, and finally dementia. During the course of development of the psychical symptoms, vertigo, severe headache, and attacks of obstinate wakefulness appear, and there are also now and then apoplectiform attacks, followed by hemiplegia. Peculiar alterations occur in the speech and voice. The speech has the slow, jerking movement as in scanning, and becomes less and less distinct. The tongue and lips and the muscles of the palate and pharynx become paretic, and hence mastication and swallowing are difficult. The ocular muscles being similarly affected, there are diplopia, or double vision, nystagmus, and amblyopia, proceeding ultimately to amaurosis.

Course, Duration, and Termination.—Not all cases pursue the typical course just described. The cerebral symptoms may be in excess, and the spinal less pronounced (cerebral sclerosis) and *vice versa* (spinal sclerosis). As Erb has well said, "the correctness of this division has not, however, been demonstrated with satisfactory clearness." Charcot has divided the disease into three parts (p. 210): the first extending from the inception to the permanent contractures—a period of very variable duration, but lasting from two to six years; the second period, in which the motor functions are almost abolished, the mind disordered, but the nutrition continues good, in which the individual is reduced to a merely vegetative existence, continues not less than four and often more than six years; the third period is comparatively brief, in which nutrition fails, digestion becomes disordered, swallowing increasingly difficult, cystitis arises from paralysis of the bladder, bed-sores form, respiration and circulation become irregular and disordered, by reason of extension of the sclerosis to the medulla, apoplectic attacks occur, and not unfrequently some intercurrent disease appears. The whole duration of the disease varies from one or two years to twenty, but the average is five to ten years. The termination may be by exhaustion or by apoplexy, but usually some pulmonary disease ends life. The termination by death is the only one known. Sometimes remissions occur that are very illusory.

Diagnosis.—The fully developed disease is so remarkable, by reason of the multiplicity of the symptoms, that a diagnosis is made without difficulty. But in the partial cases there may be much difficulty. Cerebro-spinal sclerosis is often confounded with paralysis agitans. The former occurs in youth and early manhood, the latter in old age; the former is accompanied by tremors that do not occur when the patient is at rest, and increase by volitional effort; the latter by tremors that continue during rest, and that are lessened by an effort of the will. In the former, paresis or paralysis precedes tremor; in the latter, succeeds, and long after. In the former, peculiar defects of speech, of vision, of motility, etc., occur; in the latter not. Cerebro-spinal sclerosis may be confounded with locomotor ataxia, as in both there are ataxic disorders. In the former, there are mental disorders, paralysis, contractures, tremor, troubles of speech, and preserved and increased tendon reflexes; in the latter, none of these, and ataxia without paralyses or contractures, pains, peculiar sexual disorders, and no tendon reflexes.

Treatment.—Several remedies have appeared to act beneficially, although no cures have occurred. "Marked improvement set in under the use of subcutaneous injections of arsenic," says Erb, in one case. The galvanic current has appeared to benefit in a few instances. In other cases good results, if temporary, have been produced by nitrate of silver. Hammond thinks the chloride of barium does good. The most promising treatment is the combined use of galvanism, cold hydrotherapeutic applications, carefully made, cod-liver oil and nitrate of silver; but still more useful, according to the author's experience, is the chloride of gold and sodium, and with this may be advantageously given, for a time, corrosive sublimate in minute doses.

DEMENTIA PARALYTICA—PROGRESSIVE GENERAL PARALYSIS.

Definition.—By *dementia paralytica* is meant an atrophic change in the brain characterized by a peculiar form of mental derangement, associated with general paralysis.

Causes.—The cases largely preponderate in the male sex, the disproportion being nearly four to one. The most active and vigorous period in life—from twenty-five to forty-five—is the period for the appearance of this disease. Heredity seems to be an important cause, but the data do not exist for an exact statement. Excesses—the combined effect of overwork, alcoholic abuse, and venereal indulgence—are the most influential of all factors operating to produce the disease.

Pathological Anatomy.—A diminution in the weight and volume of the brain, due to an atrophy of its gray and white substance, is the characteristic alteration in this disease. The pia mater is œdematous, generally, or in the sulci, and a good deal of water is found between the parietal and occipital lobes; the ventricles, especially the cornua, are dilated, the ependyma thickened and roughened by granular deposition; the convolutions are shrunken, particularly those of the posterior lobes, and the white and gray matter thinned and atrophic. The pia mater is greatly changed in structure, especially in the neighborhood of the vessels, and thickened by spots and patches of exudation of a yellowish color, and is readily stripped from the brain-substance. The dura mater is also much altered, closely united to the skull, thickened by exudations, and sometimes covered by a sanguineous extravasation. A peculiar change takes place in the vessels, of which the initial alteration is an increase of the nuclei in their tunics, and filling of the perivascular lymph-spaces with white and red corpuscles. The walls of the vessels become fatty or undergo the colloid degeneration. The ganglion-cells of the gray matter pass through atrophic changes, resulting in their final destruction. The membranes of the spinal cord undergo similar changes to the cerebral, but less frequently. Important alterations take place in the spinal cord ; gelatiniform degeneration, with entire disappearance of the proper anatomical elements, is the final result. The posterior columns are altered throughout their whole extent in the dorsal and lumbar portion, but in the cervical the change is chiefly in Goll's columns. Another kind of change which takes place in the postero-lateral columns is a granular myelitis, followed by hyperplasia of the connective tissue. Both kinds of change may exist together. The granular myelitis is not limited to the cord proper, but extends to the medulla, pons, and crura cerebri. The posterior roots are affected with the posterior columns, but the peripheral nerves are seldom diseased.

Symptoms.—The symptoms of this disease are naturally divisible into two groups—mental and motor derangements. A correct appreciation of the mental phenomena in these cases is of the highest importance, owing to the serious complications often arising out of the conduct of these subjects. The motor disturbances may precede, but they more usually follow, the first evidences of mental aberration. Changes in the character, disposition, and habits, and irritability and a quarrelsome disposition, quite at variance with the previous character, become manifest. Headache, transient vertigo, and inequality of the pupils, are among the early symptoms. It is observed that they fail in memory, especially of recent events ; they are absent-minded and talk to themselves. Some trembling of the lips may be seen, as well as of the muscles of the face and of the tongue. The speech becomes thick and rather guttural and is hesitating, and at the same time the voice is changed : it is nasal, and has assumed a different quality, the tenor voice becoming bass. Owing to the paresis and fibrillary trembling of the muscles of the tongue, and paresis of the muscles of the lips, the labials are pronounced with difficulty or slurred over. They early have expansive ideas and most deluded notions of what they can accomplish. Before their mental unsoundness is patent, they make purchases, or engage in ruinous enterprises, always on a large scale, and they often exhibit a marvelous ingenuity in accounting for their acts. Hence the frequent litigation growing out of the acts of such paralytics before their real condition is known. After a time their ideas become so extravagant that the least informed can understand their state. Such a man has written an immortal work, or made a great invention, will build a house many miles high, will run a railroad to the moon, possesses countless wealth, is a king, has astonishing personal prowess, has the strength of a thousand men, etc. So quick is he to forget his statements that, if exposed in an absurdity, he immediately reaffirms it in a still stronger form. He is therefore perfectly happy in the midst of his delusions of personal importance. Meanwhile he has become indifferent to all the obligations and duties of life, ceases to have any affection for the members of his family, or cares for one only, pays no attention to his affairs, and steals, without a thought of the offense. Not all cases present the evidence of exaltation of ideas and happiness from a false conception of personal importance and well-being. Some are dejected and melancholy, but the ideas of depression have corresponding vastness, and their misfortunes are the greatest the world has ever seen. During the course of development of the mental symptoms, some of these subjects are given to paroxysms of rage as blind and ungovernable as those of an epileptic. Enraged by the least opposition, or excited by some trivial incident, they will commit a murderous assault on their best friends, and this, too, stealthily and without warning. During this state there is wild excitement like acute mania. This condition of excitement may persist until death by maniacal exhaustion, or it may pass into the condition of dementia. As these attacks of excitement are accompanied by elevated temperature, it is probable they are induced by chronic meningitis, traces of which are always seen in the anatomical changes. The ideas of exaltation and of melancholy often are present in the same case, and alternate, the patient passing quickly from one to the other. Delusions are not always present. There may be a gradual and progressive failure of intelligence to dementia, without there being any delusion, unless the expansive notions, which are apt to appear some time, are so regarded. A very characteristic mental state is the unconsciousness of weakness and of disease exhibited by these subjects, unless, as may happen during a remission, the patient recovers sufficient memory and judgment to appreciate his changed state. During the height of the symptoms, although paralyzed, he has the strength of a giant, and, though suffering from ailments which in the ordinary state of the mind cause great distress, he experiences nothing but an extravagant sense of well-being. In the motor sphere very important symptoms arise. Disorders of coördination begin in the inferior extremities—an ataxic gait, reeling on closing the eyes, etc., and after a time extend to the superior extremities. Early the handwriting assumes an irregular, trembling, jerking character, and at length becomes impossible. The resemblance to locomotor ataxia is all the stronger, since there may be ocular troubles, double vision, amblyopia, and even amaurosis, altered sensations, anæsthetic tracts, etc., about the body, and retention or incontinence of urine and fæces. These locomotor ataxia symptoms, we may assume with propriety, result from the sclerotic nodules deposited in the posterior columns, but a granular myelitis attacks the lateral columns in a smaller proportion of cases, when there will occur the peculiar shuffling and helpless gait and the anæsthesia belonging to this lesion. A paretic, ultimately paralytic state of the facial nerve occurs in many cases, and the muscular system generally is thus affected. Hemiplegia, usually transient as regards the motor functions, is often the result of an apoplectic seizure which may inaugurate the symptoms, or occur at any period during the course of the disease. Instead of motor hemiplegia, sensory hemiplegia may result from a sudden attack with loss of consciousness. Although such motor and sensory symptoms disappear very quickly, the mental condition is always much injured by these attacks.* During the course of the disease, epileptiform seizures also occur; they may be unilateral or general, severe or mild. Epilepsia mitior, *petit mal*, with loss of consciousness, but no convulsive phenomena, may be substituted for the severe attacks or occur with them. Death may happen in the coma which follows an attack, or a decided remission in the symptoms, with apparent improvement in the mental state, may occur.

Course, Duration, and Termination.—Dementia paralytica is a chronic disease, but its duration can not be fixed very accurately, owing to the uncertainty which attends the time of the initial symptoms. It may be said that the cases vary in duration from one to ten years. It is true, deaths have been reported as occurring within a year, or in a few months, but there must be doubts in regard to the diagnosis in such cases. When the disease begins by apoplectic phenomena, the progress may be more rapid; and, when such attacks occur during the height of the malady, the progress downward is accelerated, although the injury caused by the apoplexy is largely recovered from. The usual course is a gradual increase in the paresis; the countenance becomes more blank, expressionless, and the muscles more relaxed; irregular jactitations occur in the facial muscles whenever speech is attempted or emotions are felt; the mode of speech becomes more and more stammering, and, as the memory becomes more and more deficient, words are omitted so extensively that the speech is unintelligible. The voluntary efforts are so enfeebled that no movements can be undertaken, and hence the patient sits motionless, or is finally bedridden, passing his urine and fæces involuntarily. Toward the end the nutrition fails, the body wastes, and an extreme emaciation is the result; rarely the face is full and flabby, the abdomen prominent.

The tongue becomes more and more paretic, swallowing increasingly difficult, and particles of food drop into the larynx, exciting suffocative attacks. Death may be caused by a pneumonia thus excited, or may occur by an apoplectic seizure, or in the coma succeeding a fit, or may be due to the exhaustion resulting from bed-sores. A considerable proportion are carried off by phthisis. It occasionally happens that a remarkable remission takes place in the condition of the general paralytic when it seems hopeless. The speech improves, the paresis of the muscular system disappears, and normal strength is restored, reason and judgment return again, and hallucinations and illusions no longer occur. This remission may last a short time, the disease revive, and the progress into its worst phases be again very rapid. On the other hand, the remission may pass on to complete restoration, the patient being restored to his friends and his work in life. This fortunate result is extremely uncommon, but has occurred often enough to require the utmost circumspection in giving an opinion. Except these cases, there is little to encourage in the course and results of this melancholy disease.

Diagnosis.—The differentiation of dementia paralytica is easily decided when the symptomatology is complete. The expansive ideas, the paralysis, the failure of memory, the lack of all moral feelings, sufficiently indicate the nature of the malady; but the cases not fully developed may be recognized with difficulty. The defects of speech, of intelligence, and the existence of paralyses with ataxic symptoms, serve to distinguish dementia paralytica from posterior spinal sclerosis. From senile dementia the differentiation is made by reference to the expansive ideas, the moral state, the peculiar affection of speech, the existence of ataxia and paralyses, and the age at which the phenomena became manifest.

Treatment.—Above all other cases, if we except acute mania, and the suicidal, there are none needing more the restraint of asylum treatment. In the attempt to put them into the asylum early, serious difficulties are encountered; for they are very plausible, and easily obtain legal assistance. Above all things, these subjects require rest, both of body and mind, and careful alimentation. The most suitable remedies are lactophosphate of lime and cod-liver oil, with quinine and morphine, to improve the nutrition of the brain and to obtain repose at night. To quiet restlessness and procure sleep, hyoscyamine ($\frac{1}{30}$ to $\frac{1}{10}$ grain) has been used with excellent effect hypodermatically. Chloral and morphine are often indispensable for this purpose, and in considerable doses. Entire rest, sufficient sleep, and a nutritious and careful alimentation, offer the best prospects of affording relief in this disease. Good results, of a temporary character, have been obtained from physostigma, and lately Ginna reports that full doses of ergot have proved remarkably effective; but it is obvious that only in an early stage of the disease could any real improvement be accomplished.

SYPHILIS OF THE NERVOUS SYSTEM.

Definition.—By *syphilis of the nervous system* is meant deposits of the secondary and tertiary stages, so called, in the meninges, in the substance of the brain and cord, and in the peripheral nerves.

Causes.—The nervous system is affected coincidently with the other viscera. The disease, pursuing its regular course, attacks the skin and mucous membrane, then the deeper organs and tissues. There is no fixed period for the appearance of syphilitic deposits in the nervous system. Susceptibility increases the rate of diffusion of the poison, and as there may be variations in its intensity, so there may be considerable differences in the time when the viscera are reached. It may be stated, in general, that the infection of the nervous system takes place during the later secondary or early tertiary period—in from one to three years usually; but it may occur within one year, or be postponed twenty years. In a large number of cases—the author has seen several—the nervous is the only secondary affection; but usually other lesions have existed, and in one third relapses have occurred. The disposition of syphilis to attack a particular part may be determined by existing injury or disease, or hereditary or acquired tendency to disease; and this is true of syphilis of the nervous system. All the causes, therefore, that tend to bring about disorders in the nervous system will determine attacks of syphiloma.

CEREBRAL SYPHILIS.

Pathological Anatomy.—The syphilitic masses, known as *gummata*, form in the subarachnoid space, or on the inner surface of the dura, and grow toward the brain. There is also a syphilitic pachymeningitis, which occurs at the convex surface of the hemispheres, especially forward on the anterior lobes, and in the middle fossa about the *sella turcica*. It is the external form, and is usually associated with bony lesions, and with the two forms of gummata. These, springing from the inner surface of the dura and from the subarachnoid space, are the most important of the syphilitic new formations. The first variety of gummata consists of a soft, reddish, translucent mass, composed of round cells and nuclei, spindle and stellate cells, distributed through the tissue of the part; and hence the density of the resulting mass is determined by the character of the tissue in which these cells are deposited. A number of cells may be closely packed in a considerable interspace, forming an alveolar arrangement, or, exuded into a reticulated tissue, will have a corresponding appearance. The new tissue contains capillary blood-vessels, and there may be extravasations by their rupture. This form is not separated by a sharp boundary from the normal tissue, but the cells push out into their surroundings. The other form of gumma is not so soft and translucent, but is dry, firm, and yellowish, so that it is sometimes said to be fatty, but is really a cheesy transformation. They exist in two forms: as a diffuse infiltration, and in circumscribed, well-defined masses, varying in size from a pea to a pigeon's-egg. A favorite site of this gumma is inclosed between the two layers of the dura, where it may attain considerable size. When the gummata form at the convexity, it is found that the granulation-tissue has completely united and blended the membranes, so that they are not distinguishable. Here the yellow masses may lie imbedded in the grayish-red gumma, and about the mass, the brain-substance into which the neoplasm projects, is in a state of white or red softening. At the base the gummata, developing, fill in all the interstices around the chiasm, the crura, and the pons. Here the grayish-red growth is chiefly seen. By developing into the adjacent brain-substance, it causes softening. A syphilitic new formation also occurs in the vessels of the base. The affected vessel is thickened, grayish, and hard, by the deposits which form a cylinder; the lumen of the vessel is encroached on, so that it transmits only one half or one fourth the usual quantity of blood. When this change occurs in several of the vessels, the cerebral circulation is much embarrassed. It will suffice to say that the changes consist in the formation of granulation-tissue in the tunics of the vessel, the morbid process beginning in the intima. Besides the gummata, the meninges may be affected by a syphilitic inflammation, which consists in the formation of thick and rather tough patches, which do not differ in structure from the gummata. Inflammation may also take place in the brain-substance, and terminate in softening.

Symptoms.—The first symptom is headache; it is usually very severe, and has this peculiarity, that it is much worse at night, and may indeed be felt only at night. The pain may disappear spontaneously, to return again, sometimes after a brief and sometimes after a long interval, but is usually continuous;* it is increased by a slight tap on the head, and its position may indicate the seat of the lesion (Lancereaux). The severe nocturnal pain causes wakefulness, but this symptom may be present when there is no pain. Vertigo, confusion of mind, irritability, inability to apply the mind to any subject, and melancholy, with suicidal feelings, are symptoms experienced with more or less severity from the time when the new formations begin to develop, and may be due to congestion as supposed by Lancereaux, but also to compression of the intra-cranial contents. After a time, fainting-attacks occur without any special cause; weakness is experienced in the legs, which give way unexpectedly; there may be defects of speech from inability to articulate; loss of the memory for words, exceeding slowness of speech; dimness of vision (amblyopia), with double vision, unequal pupils, strabismus, the ophthalmoscopic examination showing swollen disks, distended and tortuous vessels, etc.; noises in the ears and dullness of hearing; there may be maniacal symptoms, but more frequently the kind of mental defects mentioned above; epileptiform attacks succeeding to the fainting, and they may be partial, limited to one extremity, without loss of consciousness, or general, with unconsciousness. There may be, and usually are in basal deposits, especially those situated in the middle fossa, the usual site, defects of coördination, unstable gait, excessive vertigo, nausea, and vomiting, rapid impairment of vision, swollen eyelids, bleeding at the nose, etc. There are other motor defects, besides the impaired coördination and reeling gait: paresis of the muscles of one side, including the face, coming on slowly without an apoplectic seizure; there may be a mere weakness of one extremity, dragging of the foot a little, inefficient use of an arm, but still preservation of its motions, or it may be limited to one side of the face. In many cases there are, besides the motor disorders, bilateral affections of sensibility; there may be neuralgia (tic-douloureux or sciatica), but more frequently the sensations are depressed —there are extensive tracts on both sides, of complete loss of the sense of pain (analgesia) and of the sense of touch (anæsthesia), which, again, in other cases, may be more or less perfectly preserved.

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* Lancereaux, "Treatise on Syphilis," Sydenham Society edition, vol. xi, p. 46.

There is another group of cases in which, preceded by the symptoms which announce the growth of the new formations, but which may, however, be not very decided in their manifestation, there occur sudden apoplectic seizures, varying in severity from profound unconsciousness to a momentary dazed feeling, after which a hemiplegia is found to exist (Huebner). These attacks with the resulting lesions may proceed in the usual way of a hemiplegia from intracranial hæmorrhage or other lesion, of course very much influenced by the treatment, but in a certain proportion of the cases they lie in a somnolent or partly somnolent condition, from which they may be awakened, but at once lapse back. These attacks are usually preceded by headache, by a feeling of exhaustion, and by a stupid, inactive mental state, which may pass slowly into the condition of somnolence. During this state, acts are performed like those of a somnambulist, as in getting up to urinate, etc.; and when roused they awaken, gaping and yawning, but coherent, yet soon lapse back into stupor, with an air of protest at having been disturbed. These periods of somnolence vary in duration; usually continue from night to the following afternoon, and, as in a case lately seen by the author, the usual times of sleep are disturbed by severe nocturnal headache. Often, but not always, these somnolent periods are accompanied by fever of a remittent type. The somnolent period may last a few days, even several weeks, and may proceed to deeper coma ending in death, or the stupor may grow less dense, the intervals of wakefulness longer, and ultimately the somnolence disappears entirely. Cerebral syphiloma manifests itself by still another group of symptoms, namely, those of dementia paralytica. It begins with various symptoms of irritation in the intellectual sphere—confusion of mind, irritability, melancholy of an expansive kind, and ideas of grandeur. These symptoms may appear and disappear, and long intervals elapse, until at length symptoms of weakness come on, with such abnormal sensations as numbness, tingling, and formication, followed by inability for any considerable exertion, incoördination of movements, paralyses. The mental condition ultimately is that of dementia.

Course, Duration, and Termination.—There are no maladies in which the results of treatment are more conspicuous for good, and which are more influenced in their course, duration, and termination. The second form of Huebner, characterized by the apoplectic phenomena, followed by hemiplegia, is the shortest in duration, the lesions being chiefly in the vessels. Even if a cure does not take place, improvement may be effected, and the duration not be less than four years. In the second form, the opportunities for successful treatment are numerous, and the results under an appropriate medication very striking. Without treatment, weeks and months may pass before the final result is reached. The form, so like dementia paralytica, is more protracted, is subject to great fluctuations, and may continue for several years. Notwithstanding the curability of many cases—those, for example, with hemiplegia, or local paralyses, and with repeated epileptiform seizures—yet many cases resist the best-directed efforts, and for reasons that are obvious: the gummata, by pressure, produce softening and destruction of nerve-tissue, which can not be replaced. Furthermore, syphilitic cerebral affections manifest a great tendency to relapse after apparent cure.

SPINAL SYPHILIS.

Pathological Anatomy.—As in the brain, gummata spring from the internal surface of the dura, grow into the nervous matter, and unite the membranes in a compact mass. They have the structural peculiarities of gummata in the brain and elsewhere (Moxon *). Softening of the cord is a result of the presence of these new formations; partly due to pressure and partly to development inwardly of the neoplasm. Syphilitic disease occurs in the bones of the vertebræ, in the connective tissue, and in the outer layer of the dura, producing the symptoms of compression.

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* "On Syphilitic Disease of the Spine," "Guy's Hospital Reports," vol. xvi, 1870.

Symptoms.—Long after, often many years after, the specific local lesion, deposits occur in the spinal canal. According to the author's observation* the spinal troubles may be coincident with the development of fresh tertiary symptoms elsewhere. The most constant symptom is a deep-seated pain in the dorsal or lumbar region, increasing at night; a pain of such severity as to require powerful anodynes to obtain sufficient relief for sleep. There may or may not be tenderness on pressure. Usually a great deal of pain is experienced in one or both of the sciatic nerves, and tingling, numbness, and burning sensations in the legs and feet. More or less weakness, a strong sense of fatigue on slight exertion, stiffness and cramps are experienced in the muscles of the spine, of the neck, and of the extremities. As the disease is developing, the general system sympathizes to a remarkable extent; a peculiar earthy hue of the face, emaciation, and debility are observed. The symptoms may continue at this point for a long time, or partial improvement take place, and then, after some weeks or months of inaction, more serious symptoms come on. When the symptoms become active again, paralysis begins and proceeds with great rapidity, and becomes so complete that not a toe is movable. The paralysis may be due to disease of the dorso-lumbar enlargement, and both lower limbs be completely paralyzed (paraplegia) as to motion, sensation, and the reflexes. The sphincters will also be involved, and incontinence be added to the other troubles. There may be partial paralysis, one limb involved. When the arms are affected, there will be oculo-pupillary phenomena, and the respiratory muscles will be paretic or paralyzed if the disease is high up in the cervical region. These spinal troubles of syphilitic origin may be associated with corresponding cerebral lesions, when, of course, the symptoms will partake of both. There is a form of acute spinal paralysis described by Huebner which comes on during the first secondary symptoms, and is characterized by a sudden paraplegia or paralysis of one arm and the opposite leg. In a few hours, or a day or two, the mischief is wrought, and the paralysis complete.

Course, Duration, and Termination.—The course of the principal forms of spinal lesions is very protracted, and they appear long after the local primary. Rightly treated they get well promptly, but, as is the case with the cerebral disease, they are prone to relapse, yet the ultimate cure is probable. When paraplegia has occurred with absolute paralysis, a cure may often be effected in a few weeks; but that this favorable termination shall take place it is essential that the injury be recent. If the cord has been damaged, permanent disability will remain, although the disease may be arrested. Old cases may terminate fatally by exhaustion from cystitis and bed-sores. The acute form, described by Huebner, seems to be very unmanageable, and to reach a fatal termination by extension upward. In the spinal as in the cerebral form, much depends on the treatment instituted.

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* "On Syphilis of the Nervous System," "The Clinic," 1874.

SYPHILIS OF THE NERVES.

Pathological Anatomy.—The cerebral nerves seem to be chiefly if not the only nerves attacked by syphilis. The deposits may be exterior, and press on the nerve-trunks, producing a neuritis, which leads to atrophic changes and degeneration. A gumma surrounding a nerve-trunk unprovided with a sheath will grow into the tissues of the nerve, and syphilitic granulation-tissue may be deposited in places, and develop in the ordinary way.

Symptoms.—The results of such affections of nerve-trunks have a different expression according to the function of the nerve. Irritation of a sensory nerve produces pain in its peripheral distribution ; but, if the nerve is destroyed, anæsthesia and analgesia are experienced. On the other hand, if a motor nerve is irritated, spasms or tonic contraction will ensue in the muscles to which this nerve is distributed ; if the nerve is destroyed, paralysis ensues. As the cerebral nerves are usually affected, the same symptoms result from syphilitic neoplasms as have been described in connection with other neoplasms or tumors of the brain.

Diagnosis of Syphiloma of the Nervous System.—The first point to determine is the occurrence of syphilitic infection. The peculiarities of the syphilitic affections of the brain are their diffusion, the irregularity in the development of the symptoms, the simultaneous existence of irritation and depression, the periods of spontaneous improvement, the remarkable change in the condition of a patient receiving iodide of potassium or mercury in some form, etc.

Treatment.—In these affections the most marvelous change is wrought by sufficient doses of the iodide of potassium. No time is to be lost in its administration, and usually the largest doses are required. Sometimes mercury does better, and lesions do not yield until it is administered. It has been observed, also, that cases not yielding readily to specific treatment, will do so on the exhibition of pilocarpine to promote absorption.

CEREBRO-SPINAL NEUROSES.

EPILEPSY.

Definition.—By the term *epilepsy*, as here employed, is meant true or essential epilepsy, and not eclampsia, nor convulsion from such cause as tumor, abscess, etc., of the brain.

Causes.—Heredity occupies the first place as an etiological factor. In Echeverria's[*] cases, about twenty-five per cent., and in Reynolds's[†] about thirty per cent. were distinctly due to hereditary transmission. It is a neuropathic constitution or tendency which is inherited, and this exhibits itself in various forms in different generations. In one generation it is neuralgia, nervousness, paralysis; in another, epilepsy; in a third, insanity. Next to the inheritance of a neurotic tendency, in point of importance as a cause, is the influence of drunkenness in the father on the product of conception. Sexual excesses and onanism are held to be frequent causes, but much exaggeration has existed in regard to their effects in this way. They are more frequently the result than the causes of epilepsy. As regards age, the greatest number of cases occur in the decennary from seven to seventeen. As regards sex, the two are about equal in their liability to the disease. According to Reynolds, not one case which was hereditary began after twenty, while twenty-six per cent. of those not hereditary were affected after twenty. Irritation of peripheric nerves, dentition, and injuries to the cranium, are among the occasional causes. Epileptic seizures have been excited by various psychical impressions—by fear, by irritation, by chagrin, and other powerful emotions.

Pathological Anatomy.—There is no morbid alteration peculiar to epilepsy. In this important respect true epilepsy differs from epileptiform seizures. Although there is no special change, various accidental pathological alterations are found in the cranial cavity. Changes in the contour and structure of the skull; thickened, indurated, and calcareous meninges; increase in weight of the brain according to some (Echeverria), and diminution of weight according to others; changes in the hippocampus (Meynert); tumors of the cortex; variations in the distribution of the gray matter—are gross lesions which have been ascertained to exist in old cases of epilepsy. Long ago Schroeder Van der Kolk[‡] found alterations in the medulla, consisting in dilatation of the arterioles and fatty degeneration of their tunics. Echeverria[§] confirmed these observations and added investigations of his own, to the effect that not only are the vessels enlarged, their tunics fatty, but that hyperplasia of the neuroglia and atrophy of the cells of the medulla are constant changes in epilepsy. The same author has ascertained the existence of sclerotic changes in the ganglia of the sympathetic, but the relation which such changes bear to the production of epilepsy is by no means clear.

Symptoms.—The phenomena of epilepsy are exhibited in two forms of seizures, and in the state of the affected individual in the interval between the convulsive or unconscious attacks. The epileptic seizures are: *epilepsia gravior*, the severer epilepsy, the epileptic fit, called by the French writers *le grand mal*, and *epilepsia mitior*, milder epilepsy, *le petit mal.* Adopting the classification of Jaccoud, we have the first form occurring in two modes—the *common* or *ordinary* form, and the *apoplectic ;* the second also in two—*vertigo* and *absence.* Many cases of the common form begin without any indication of their approach, but a certain number are preceded by definite sensations and warnings. The term *aura* is applied to a singular phenomenon preceding the attack and indicating its approach. No longer used, in accordance with its original signification, as a *breath*, this term expresses any manifestation, sensory, motor, or psychical, which gives warning of a paroxysm: it may be the sensation of a breath, the flowing of a hot or cold liquid, numbness, tingling, even a severe pain passing with great rapidity from the periphery to the brain. Again, the aura may consist of an impression on an organ of sense, as a flash of light, a strange odor, or a rumbling noise in the ear; or in some local muscular spasm or cramp; or some specter or other hallucination rising up in the mind. Warnings may be more remote, occurring some days before the seizure, when they take the shape of mental or moral perturbation; sadness and despondency of mind, a gloomy reticence and suspicion are experienced, or an excited, irritable, quarrelsome, even dangerous and malignant state of mind comes on. More frequently than these symptoms occur merely headache, dizziness, and some confusion of mind, for a few hours or a day or two before the seizure. In a large proportion of cases seen by the author, the prodromal symptoms consisted in a sense of præcordial oppression, epigastric uneasiness, and nausea, the attack following immediately on the rise of a peculiar sensation from the epigastrium to the brain.* With or without an aura, the epileptic paroxysm when it occurs is sudden. It consists of four distinct acts: a sudden fall; loss of consciousness, with pallor of the face; a peculiar cry; general convulsions. In any situation or place the individual attacked happens to be, he falls—down the stairway, into the fire, against an article of furniture; or if, mercifully warned by some sensation, he has the opportunity, he places himself in a position of safety. The fall may be to one particular side, on which scars will be found to indicate the direction taken in falling. The fall occurs because loss of consciousness supervenes, and the control is at once withdrawn from the voluntary muscular system. Sensibility, motility, perceptions, the special senses, the reflex functions even, are at once and entirely abolished. The face grows deadly pale, and this is due to a sudden spasm of the arterioles of the head, whence the amount of blood passing to the brain is greatly reduced. At the moment that unconsciousness takes place, a peculiar cry is uttered, "shrill and terrifying to man and beasts," is the description of Romberg.* It may be a mere groan, and there may be an entire absence of all sound. Immediately on the occurrence of pallor of the face, the muscles of the body generally assume a position of tetanic rigidity; the head is drawn back or to one side, where it is firmly held; the jaws are tightly closed, the lips retracted into the sardonic grin, the eye fixed in a stern expression, the brow corrugated; the fingers and toes are extended, widely separated; the respiratory muscles similarly tetanized, respiration is suspended; the pulse is small, firm, and variable in frequency; a rapid venous stasis, cyanosis of the face, and blueness of the lips succeed to the momentary deathly pallor, because of the arrest of respiration and compression of the great venous trunks by the rigid cervical muscles. Just as the *tetanic stage* begins, a loud, strong, and protracted whistling inspiration is made, and then ensues the rigidity of the respiratory muscles. The tetanic condition may not be universal, may be limited to a few muscles, as those of the head and eyes, the spasms being clonic at the outset; or there may be no rigidity, the muscular twitching beginning at once, or, on the other hand, there may be nothing more than transient rigidity of the voluntary muscles. This rigid stage lasts from a minute to a minute and a half, and is succeeded by the stage of *clonic convulsions.* At first the muscles of the face, lips, tongue, pharynx, and larynx begin to twitch, the face to make horrible grimaces, the eyes to roll in their sockets. The face is still blue, the lips blue, but, as respiration goes on, the blueness is mixed with red, the superficial veins are swollen, the lips are extruded with each expiration and are covered with froth, often with bloody froth, the breath issues with a whistling, stridulous noise, the inspiration being labored, loud, sonorous, the teeth grind together, and often the cheek or tongue is caught and chewed, thus furnishing the blood which is mixed with the froth. The muscles of the extremities are violently agitated, thrown about, and with such violence that severe injuries are sustained, even fractures of the long bones or dislocations. Vessels give way and ecchymoses of greater or less extent are formed about the eyelids, and in the mucous membrane of the tongue and lips. By these marks may be ascertained the existence of nocturnal epilepsy, which otherwise remains undiscovered. The clonic stage lasts one, two, or three minutes, and its close is announced by the subsidence of the convulsions; they occur less and less, and at length there is only an occasional twitch of the muscles about the mouth, and presently all is still, the individual passing into deep sleep, in which the iris, before dilated, contracts, the respirations become regular, deep, and full, the muscular system relaxed, and the skin warm and perspiring. There may be, indeed, a condition of coma lasting several hours after the convulsive stage, and fecal and seminal discharges may occur involuntarily. The duration of the comatose stage varies from a few minutes to several hours, and the patient rouses with a rather surprised, or dazed, or sheepish expression, and is entirely ignorant of the affair through which he has just passed, unless the bitten tongue or cheek reminds him of former experiences. Usually the effect on the mental and moral state is that of improvement, and the patient feels better than before. Attacks may succeed to attacks. Without coming out of the condition of coma, another convulsion succeeds to the previous one. In other cases the recovery from each paroxysm is complete, and the convulsions occur with a distinct interval of an entirely normal state. The number of paroxysms during a period of twenty-four hours may be from one to fifty—even more. Immediately succeeding the convulsions in some epileptics, there occur attacks of delirium or hallucinations, or they pass into an excitable, quarrelsome state, and are prone to commit homicidal acts. Physicians have frequently to testify as to the mental condition of epileptics, on trial for acts committed in the mania which succeeds to convulsions.

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[*] "On Epilepsy," by M. Gonzales Echeverria, M. D., New York, 1870.

[†] "Epilepsy," etc., by J. Russell Reynolds, 1861, p. 123.

[‡] "On the Minute Structure and Functions of the Spinal Cord," Sydenham Society edition, 1859, p. 231, *et seq.*

[§] *Op. cit.*, chapter xi, p. 46.

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* Gowers ("Gulstonian Lectures," "Lancet," March 20, 1880) says this sensation occurred in one half of his cases.

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* "On Nervous Diseases," "Sydenham Society's translation," by Sieveking.

The apoplectic form of Jaccoud differs from the ordinary form just described, by the depth and duration of the succeeding stage of coma, by the evidences of cerebral congestion present, and by the paralysis—temporary or more permanent—usually in the form of hemiplegia, succeeding to the clonic convulsions. The second form—*epilepsia mitior*, milder epilepsy, or *petit mal*—exhibits iself in the two forms of *vertigo*, or vertiginous sensations, and *absence* or instantaneous unconsciousness in the sphere of ideation. In the former, the patient is seized with a severe vertigo, in which all surrounding objects are in motion, and he is unable to maintain the upright position, and would fall if not supported. With the vertigo there is loss of consciousness lasting for a second, when the normal state is restored. Usually, the vertiginous sensations and the loss of consciousness are accompanied by some partial convulsive phenomena; as grimaces, twitching of the muscles of the face, grinding of the teeth, movements of rotation of a member—of the arm, for example —or of the whole body, running forward suddenly. On an instant consciousness returns, the patient looks around with a foolish expression, it may be, and the attack is over. By *absence* is meant *absence of mind*, but not in the popular sense—in the technical sense, in this connection, of total abolition of ideation, for an instant of time. The attack may occur at any time, and consists in the most transient suspension of consciousness—in the midst of a sentence, sewing, walking, or writing: for the instant all thought is suspended;

the sentence being uttered, the sewing, the walking, or the writing is stopped, and then immediately resumed, so that the brief gap may attract no attention. Observing the appearance of the individual thus attacked, there will be seen a sudden pallor of the face and dilatation of the pupil, but no other objective phenomena. These forms of *epilepsia mitior* may precede, for a long time, the fully developed attacks, or may occur with them. The popular notion of the little importance of these seizures is not justified by the results, for absence is particularly injurious to the mental faculties. In all of these forms of epilepsy the loss of consciousness is the central fact, and without it, according to many, there can be no epilepsy. There are, however, numerous examples of convulsions, partial and general, without loss of consciousness. Dr. Hughlings Jackson* defines epilepsy as "a *sudden*, *excessive*, and *rapid* discharge of gray matter of some part of the brain on the muscles." It does not necessarily involve the loss of consciousness. His notion is that any mass of gray matter may get into a highly excited state by some kinds of irritation—"reaches very high tension and very unstable equilibrium, and therefore occasionally 'explodes.'" Irritation of a part, the destruction of which causes hemiplegia, will induce unilateral convulsions of the same region. Local convulsions, as in an arm, for example, may therefore be a "discharging lesion of a small extent of irritated gray matter." There are masked or concealed epilepsies, taking the form of tic-douloureux, or neuralgia of the fifth nerve, convulsive tic, or histrionic spasm, and angina pectoris. After a time the paroxysms assuming these forms take on the proper epileptic character, or the epileptic seizure alternates with its counterfeit. Again, epilepsy may take the form of an acute delirium (Falret's *delirium epilepticum*). The peculiarity of this affection is its sudden and unaccountable appearance, and its equally prompt and unexpected disappearance. Often the delirium takes the form of an "insane impulse," in which acts of violence are committed, or of obscene and violent language, or of some senseless conduct. It may become excessively violent and destructive, leading to the performance of atrocious murders. This condition of mind is transient and disappears in a few hours or in a day or two, and the patient is either totally unconscious or has the remembrance as of a vague dream.

Course, Duration, and Termination.—Epilepsy is one of the most chronic of diseases, and its duration numbers many years. At the outset there may be many months' interval between the attacks, but, as the case progresses, the attacks increase, and the intervals between them become shorter. The periods of return are very irregular. Now and then attacks strictly antiperiodic are encountered, and others are connected with the menstrual functions. As attacks are often determined by preventable causes, the number may be much increased by indiscretions. Among these are indulgence in alcoholic fluids, sexual excesses, and errors in diet. Probably the last named is the most important of these noxious influences. Nocturnal attacks may escape recognition for a long time, and the origin of the disease dates from some diurnal attack, or from a seizure in which the bitten tongue, ecchymoses, and general muscular soreness served to indicate the nature of the disturbance. An unexpected decline in mental power, changes in the disposition, and impaired health in certain directions without any apparent reason, may be explained by nocturnal epileptic attacks. The existence of epilepsy is not incompatible with a condition of perfect health. In the interval between the attacks, still more in the future progress of the cases, various alterations in the motor, sensory, and intellectual sphere are produced. In the motor group may be mentioned clonic convulsions or clonic or tonic spasms in a single extremity, or group of muscles; in the sensory, numbness of certain areas in the extremities, headache, neuralgia, etc. The most important results of epileptic seizures are changes in the intellect, weakness of memory, impaired judgment, etc., gradually increasing until ultimately these unfortunates pass into the condition of dementia. Occasional epileptic attacks do not seem to have much influence on the condition of the mind, and in confirmation of this opinion are always quoted the cases of Cæsar, Napoleon, and Petrarch. The statistics of Reynolds prove that the number of attacks alone is not responsible for the effect on the intellect, but the mind suffers more when the attacks follow in quick succession. Epileptics early suffer changes in the moral sphere, in the affections, the disposition, and the emotions, before any intellectual decadence is observed. Although the prognosis is unfavorable as respects cure, decided amelioration can be effected in a large proportion. A few cases are cured, and the number of cures increases with the improvements in therapeutics. The earlier the treatment is undertaken the more favorable the termination. The less the number of attacks within a given period and in the aggregate, the more favorable. If there be a distinct peripheral cause, as injury to a nerve, a tape-worm, etc., the prognosis is more favorable; but, when the status epilepticus is established, it does not suffice merely to remove the cause. If central lesions exist, the termination by recovery seems quite impossible. Heredity apparently increases the intractability of the disease, but some notable exceptions have been published. Nocturnal attacks are less amenable to treatment than diurnal. The forms of epilepsia mitior are, as a rule, more difficult to manage than epilepsia gravior. Absence especially has disastrous effects on the mind. Finally, treatment has an important influence for good or evil over the course, duration, and termination of epilepsy in all its forms.

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* "A Study of Convulsions," and "On the Investigation of the Epilepsies," and various papers.

Treatment.—The success of the management of epilepsy depends largely on the skill with which various sources of peripheral irritation are investigated and removed. Every case, therefore, requires the most deliberate and searching investigation. Has there been an injury? Is it of the cranium or of a peripheral nerve? Many cases have been cured by the application of the trephine, and the number is increasing. So favorable have been the results of this practice that, if a severe blow on the cranium has been followed by epilepsy, and any injury of the bone can be detected, the trephine should be used. Cicatrices so situated as to exercise pressure on a nerve should be dissected out—a practice of special necessity when an aura or any uneasy sensation starts from the affected part. If there be a defined aura so situated as to be intercepted in its passage to the brain, various expedients have been resorted to for this purpose, as a ligature about the thigh, leg, or arm, the application of a blister to surround the limb, or the cauterization, by nitrate of silver, of a band around the extremity. Permanent relief has been obtained by cutting down on the point whence an aura proceeds, and not only removing a source of irritation, but dividing or stretching a nerve-trunk. When the impression arises at the epigastrium and passes thence to the brain—probably the most frequent of all prodromic symptoms or warnings—most careful attention must be given to the diet. The author has witnessed more good from regulation of the diet than from any mode of medication. Epileptics eat largely and bolt their food. When stomachal symptoms exist, an epileptic should be restricted to the milk-diet for several weeks, and should then gradually have additions made to it; but the permanent diet should not exceed milk, eggs, a little meat once a day, a single vegetable, a very little bread and butter, and one fruit. Restriction to this plan of diet will often effect remarkable improvement. If there be worms present in the canal, they should of course be expelled. If stomach symptoms are present, good results are obtained from drop-doses of Fowler's solution three times a day, from half-grain doses of the oxide or nitrate of silver, or a suitable quantity of oxide of zinc. These remedies are beneficial only in cases of epilepsy dependent on stomachal derangements. The danger of staining by the use of silver remedies should not be overlooked. From the negative point of view there are several important questions connected with the stomach and alimentation. Coffee, tea, tobacco in any form, and all kinds of alcoholic drinks, must be forbidden to all classes of epileptics. It is important to prevent paroxysms, since habit enters largely into the mechanism of epileptic seizures. The means of intercepting an aura have been referred to. Brown-Séquard suggests various peripheric irritations—pulling on the great-toe, inhaling a little carbonic-acid gas, etc. The inhalation of ether and chloroform may render the attacks less severe, but the practice is questionable. When the attacks are nocturnal, a sufficient dose of chloral, or better, the hypodermatic injection of morphine at bed-hour, will act most efficiently to prevent them, but as the morphine habit will be quickly formed, other remedies should be preferred. The nitrite of amyl by inhalation will often avert an impending attack. The advantage of this remedy consists in the facility with which it is employed. A perl containing three to five minims can be broken up in a handkerchief and inhaled without delay. Nothing should be done during the paroxysm but relieve the body of all constricting bands, and put the epileptic in a position where he will not injure himself. The question of a suitable remedy for the disease is by no means a complicated one. There can be no question of the superiority of the bromides, and notably the bromide of potassium, over all other remedies. Their long-continued use is attended with few disadvantages, and the mental condition improves rather than declines under their employment. The bromides of sodium and potassium are chiefly administered, but while the potassium bromide is rather more effective as a remedy, the sodium bromide is far less hurtful, and should be preferred in cases requiring the protracted administration of such remedies. The point to arrive at in the course of the use of the bromides is an anæsthetic state of the fauces—an important fact which we owe to Voisin. The fauces must have their reflex sensibility so far reduced that no movements are excited by touching the palate, base of the tongue, or any part of the throat. The amount required to produce this result will vary, according to the individual susceptibility, from one half to two drachms per day, but it should be borne in mind that it is not the quantity of the medicine required, but the effect produced, which should guide the administration. Bromism may be prevented by the occasional use of a purgative, by maintaining free action of the kidneys, and by combination with Fowler's solution, two or three drops morning and evening. Next to the bromides, probably, are strychnine and picrotoxin in utility. The author has given strychnine with the bromides in cases of epilepsy occurring in weak and anæmic subjects. It is adapted to those cases in which there is mere instability of nervous matter, due largely to anæmia, and is contra-indicated in those cases characterized by exalted reflex excitability, with peripheral irritation. In the treatment of epilepsy by bromides, the mistake is made of giving it irregularly, or of suspending it capriciously. It should not be suspended, even if bromism occur; it should be diminished in amount and active elimination set up, and then resumed in the dose necessary to maintain anæsthesia of the fauces. It should be continued for a long period after the convulsions have ceased, probably not less than two years.

Cases of nocturnal epilepsy are not so much benefited by the bromides as the diurnal. In the former the author has seen better results from picrotoxin, and from curara, especially. In nocturnal cases characterized by depression, phosphorus and phosphide of zinc have seemed to do much good. In the state entitled "absence," *petit mal*, and in epileptic vertigo, nitro-glycerine has acted favorably.

HYSTERIA.

Definition.—*Hysteria* is a functional nervous trouble, characterized by various motor, sensory, and intellectual disturbances, and by excessive variability in their seat and manifestation.

Causes.—Hysteria is almost exclusively confined to women, and only occasionally witnessed in men. The sexual condition, the social habits, the repression which a very limited sphere of activity enjoins, and a much greater mobility of the nervous system, are supposed to be the chief reasons for the relatively greater prevalence of hysteria in females. The age at which hysterical manifestations appear is not a fixed one, and, although most frequent from puberty on for ten or more years, attacks occur from childhood. In Briquet's collection of four hundred and twenty-six cases, two hundred and twenty-one appeared between the twelfth and twentieth years of life. Undoubtedly, that mobility of the nervous system, and instability, on which the manifestations of hysteria depend in the mother, are transmitted to the daughter. If the so-called neurotic type of constitution is inherited, in one generation it may assume the shape of hysteria ; in the next, epilepsy ; and in the third, insanity. But the hysterical type, as such, is more directly inheritable. That derangement of the female sexual organs—especially of the uterus and ovaries—is the essential cause of hysteria, is an opinion no longer entertained in any quarter. It can not be too strongly insisted on that there is a peculiar morbid state of the nervous system—a neurosis—either inherited or acquired, and that various kinds of disturbances may excite the morbid manifestations. These disturbances may be in the sexual system, in the digestive, in the circulatory, or in the nervous. This peculiar state of the nervous system may be acquired by faults of early training, by a lack of personal discipline, by frequent alternations of feeling, by mortification, chagrin, and other moral and emotional excitements. That hysteria may exist independently of sexual causes is quite proved by the fact that violent hysterical paroxysms occur in women congenitally deficient, and wanting in uterus, and ovaries, and all sexual characteristics. The instability of the nervous system belonging to hysteria is much increased by certain physical causes—notably by anæmia. When the blood is impoverished, the nervous tissue becomes excessively irritable, and the discharges of nervous force are frequent and irregular, while deficient in sustained force.

Pathogeny and Symptoms.—No structural alterations have been detected in the centers where the disturbances of function exist. Hence hysteria is properly a neurosis—a functional disorder. The old notion, that uterine disease is a necessary element in hysteria, as the word indicates, has long been abandoned. The first manifestations of hysteria are usually trivial—mere irritability or mobility of disposition ; rapid changes of feeling without apparent motive ; noisy and tempestuous transitions of sadness and joy, tears and laughter. In the course of development, physical are added to these merely psychical changes; quick and unaccountable alternations of cold and heat, that are purely subjective, and felt usually in the extremities; numbness, tingling, and other altered sensations, which are extremely irregular, now severe, awakening fears of paralysis, now forgotten in the presence of something interesting to occupy the attention, access of suffocative feelings, "pain around the heart," palpitations, quick breathing, a sense of fullness of the stomach, eructations of gas, and the rising of a globe to the larynx (globus hystericus), producing a sensation of choking; alternate flushing and pallor of the face; restlessness; the whole ending, it may be, in prolonged laughter, but more usually in crying, and in a profuse urinary discharge, the urine being pale and watery. Such an attack may occur, with more or less frequency, in a young woman of good health otherwise, and may never advance beyond this. In addition to the symptoms just described, there may be spasmodic phenomena, tonic and clonic. When the more severe attacks approach, they exhibit alternations of chilliness and heat, they yawn and gape a great deal, the limbs are in a condition of unrest, of "fidgets," they laugh and cry, and equally without reason, they urinate frequently, the heart palpitates, they choke with a ball rising up into the throat and gasp for breath, sobbing, and coughing with a loud, metallic clang, the jaws are fixed, the face retracted, the teeth grinding together, the hands clinched, the limbs drawn up and rigid. Such are the phenomena of the tonic convulsion. In a few minutes, usually, or in an hour or two, the attack subsides, the patient sheds a flood of tears, passes a large quantity of limpid urine, and goes to sleep exhausted. In other cases, a brief stage of tonic rigidity is succeeded by irregular clonic convulsions, the patient throws her limbs about, screams, tears at her throat to remove the choking sensation, sobs, gives forth repeated, loud hiccough, the abdomen is full of gas, and there are loud borborygmi; sometimes the pelvis is moved in a rhythmical manner, and the limbs are fixed. There is no loss of consciousness, the reflex movements of the iris and eyelids are preserved, and, although the jaws are rigid, if fluid reach the fauces it is soon swallowed, and the realization of surrounding events is preserved. As a result of the violent muscular efforts, the skin, which was at first cool, becomes warm and perspiring. These convulsions last for several minutes, or as many hours. They subside in a flood of tears, the body is completely exhausted, and the patient sinks into a deep sleep. During these attacks, usually, the reflexes are increased, and pressure on certain regions of the face, head, or spine, or on the ovaries, will increase the convulsive movements. According to Charcot, pressure on an ovary will excite attacks, and firm pressure may arrest hystero-epilepsy. In some cases there are no convulsions, but the patient passes into *ecstasy*, a condition of fixed immobility and death-like pallor of the face, half-closed eyes, almost suspended respiration, extremely feeble, hardly distinguishable pulse—an appearance of death. In other cases, the condition of ecstasy is associated with catalepsy—in which the limbs retain the position in which they are placed. The duration of the cases varies. Instead of terminating, in a certain proportion there are remissions merely, and hence the attacks may persist for several days. The critical evacuations which announce the end of the seizure do not occur in the remissions. There are no regular periods of return, except that they are more apt to be present during the menstrual periods, and do not occur at night. If the moral or mental state and the bodily conditions which favor the attacks continue in operation, a succession of seizures may be expected.

Hysteria is associated with widespread disorders in the sensory, motor, psychical, and vaso-motor systems, which appear at the onset of the disease or during the intervals between the attacks. The retina may be so sensitive to luminous impressions that the least light becomes intolerable; hence it is that so often the hysterical are found in dark apartments. Flashes of light and floating objects appear before the eyes; more complex impressions of scenes and persons are reproduced, and hallucinations are perceived. In the same degree hyperæsthesia of the auditory is present, and even a whisper causes pain, while various loud, roaring, subjective noises are heard. Sometimes a remarkable acuteness of hearing is developed, and out of this may grow conscious deceptions. The hysterical, like the insane, may hear voices, but the results differ in the important respect that the former realize their origin. The sense of smell in the hysterical is much perverted, and they are acutely sensitive to odors. Remarkable perversions of taste are also manifest. The hysterical have a propensity for eating chalk, slate-pencils, sealing-wax, etc. As regards general sensibility, there may be more or less hyperæsthesia and hyperalgesia, in particular spots or areas, and between these areas of anæsthesia. Pain is one of the most usual and widely distributed of the sensory disturbances in hysteria, and headache is the most common form. There may be general headache, with such a degree of hyperæsthesia of the scalp that combing the hair is painful. The headache may be localized to a particular point at the top of the head, or to one temple, or to the supra-orbital ridge, may be exceedingly violent, and accompanied by chilliness and feverishness, nausea, vomiting, and constipation. This form of headache has been called *clavus hystericus*. It is very apt to come on at or about the menstrual epoch. Neuralgic pains occur in the mammæ, which become irritable and tender, or in the præcordial region, which are always referred to the heart, and in the left side, about the sixth or seventh intercostal space. The last-mentioned pain is more frequently referred to than even the headache. Hysterical women suffer greatly from the evolution of gas in the intestine, and hence colics are frequent. Hyperæsthesia of the abdominal wall may also be present, and simulate peritonitis; but exquisite pain is complained of before the skin is touched, and, when the attention is withdrawn, the abdomen can be pressed upon without any flinching. Gastralgia is a very usual symptom; emptiness, abnormal fullness, boulimia, and an utter disinclination for food, are among the very contradictory sensations. The presence of a parasite and its movements are often insisted on. An irritable bladder is a common symptom. Pain in the extremity of the coccyx, or coccydinia, is complained of, usually after the first confinement, or from the results of a blow, and is a peculiarly unmanageable symptom. The much-debated spinal irritation is also an extremely frequent symptom in cases of hysteria. It consists in tenderness and pain on pressure of the spinous processes of a few vertebræ, or of the parts immediately adjacent. Spinal irritation has no more importance than any of the pains which occur in the course of hysteria. The joints are similarly affected, especially the knee, which becomes painful and swollen the more the attention is fixed on it. This affection, first described by Sir Benjamin Brodie, is known as the hysterical joint. The peculiarity of it is the occurrence of pain and swelling rather around than in the joint, but often the joint is simply rigid in a position of flexion. Extensive spots, entirely anæsthetic, occur in hysterical subjects. Analgesia may be present to such a degree that extensive injuries can be inflicted without consciousness of pain. The anæsthesia may be limited to one side—hemianæsthesia. The muscular sense and the appreciation of weight may be lost, and the senses of touch and temperature retained. Amblyopia may be the result of anæsthesia of the retina. Paralyses in the course of hysteria are numerous and perplexing. Dysphagia may exist from paralysis of the pharynx, aphonia from paralysis of the vocal cords, and both may occur on the instant, and disappear as suddenly. Paralysis of the bladder and retention of urine, requiring the catheter, is a common symptom of hysteria. Paralysis of a member, of several, or of muscular groups, known as hysterical paralysis, assumes various characters: one extremity may be affected, or one upper and one lower extremity on opposite sides; it may take the form of hemiplegia, of paraplegia, or all four extremities may be affected simultaneously. It may be partial or complete; it may come on gradually, or appear suddenly after a fit, or without any reason. The electric reaction is normal, unless the limbs are wasted from long disuse. There may be anæsthesia with the paralysis, but not necessarily, and, when that is the case, the electro-sensibility is wanting. On this Duchenne founded a distinction between hysterical and other forms of paralysis, but incorrectly so, since in some the sensibility is normal or even increased. The duration of hysterical paralysis is very variable; it may continue for a few hours, a few days, many months, or several years, and it may unexpectedly disappear from one part to attack another. With or without palsy there may be contraction, or after the paralysis has existed for some time the contraction may come on. In the upper extremity, a spasmodic flexion of the fingers, hand, or forearm may occur; in the lower, spasmodic extension of the hip, knee, and ankle-joints. The behavior of the contractions is the same as the paralysis—they continue a variable period, to be suddenly terminated by some moral influence. Various disturbances ensue in the realm of the vaso-motor nervous system—irregularity and weakness in the heart's action; amenorrhœa and dysmenorrhœa; epistaxis, hæmoptysis, and hæmatemesis; stigmatizations. As extraordinary ingenuity and perseverance and self-denial are employed to execute the deceptions by which they produce the appearance of these maladies, to excite sympathy and attention, the physician must be on his guard lest he be led into error. Remarkable mutilations and personal injuries are effected, to excite sympathy or wonder in those about them. Influenced by a morbid craving for strange excitements, an hysterical girl will injure an infant, burn a house, stick things under the skin, drink her urine to make believe that none has passed, produce pins as having come from the bladder, or draw a dead animal from the vagina, etc. Indeed, there is scarcely a limit to the extraordinary fancies or to the eccentric acts of the hysterical. Besides these perverse and singular acts, growing out of moral perversion, the hysterical may undergo forms of mental derangement, the most persistent ending their days in asylums. In some, the mental disorder takes the place of melancholia, and they tend to injure others or to the commission of suicide, to give vent to their notions of misery. In others, the disorder is in the direction of moral mania: they steal, injure articles of clothing, or set fire to the house; they are given to sexual vices, to strong drink, and are utterly without a moral sense. In others there will be developed mania with delusions, often of a religious kind.

Course, Duration, and Termination.—Beginning often at a comparatively early period, hysteria reaches its highest development from puberty to thirty-five, afterward decreasing, to disappear in old age. Those developing slowly under hereditary influence and by example are the most difficult to cure. In that admirable little book, "Fat and Blood," Mitchell describes with a master hand the course of many cases: "But no matter how it comes about, the woman grows pale and thin, eats little, or if she eats does not profit by it. Everything wearies her—to sew, to write, to read, to walk—and by and by the sofa or the bed is her only comfort. Every effort is paid for dearly, and she describes herself as aching and sore, as sleeping ill, and as needing constant stimulus and endless tonics. Then comes the mischievous *rôle* of bromides, opium, chloral, and brandy. If the case did not begin with uterine troubles, they soon appear, and are usually treated in vain if the general means employed to build up the bodily health fail, as in many of these cases they do fail. The same remark applies to the dyspepsia and constipation which further annoy the patient and embarrass the treatment. If such a person is emotional, she does not fail to become more so, and even the firmest women lose self-control at last under incessant feebleness. If no rescue comes, the fate of the woman thus disordered is at last the bed. They acquire tender spines and furnish the most lamentable examples of all the strange phenomena of hysteria." Under the influence of marriage and child-bearing, the hysterical troubles may disappear entirely or for a long period, returning from time to time, but much less severely. In most cases there are remissions and exacerbations, and those cases characterized by the most severe symptom may have the shortest duration. The danger to life is inconsiderable. The probability of mental disorder arising is slight, but the prospect of cure is, in the cases of long duration, very remote and uncertain.

Diagnosis.—The diagnosis of hysteria rests on the age, sex, the variability and diffusion of the symptoms. There is no possibility of mistaking an attack of vapors. Epilepsy is distinguished from the convulsions of hysteria in the order with which the several stages occur, in the loss of consciousness and the abolition of reflex movements, biting the tongue or cheek, the after-coma, and in the absence of hysterica phenomena in the interval. In those cases of epilepsy occurring in hysterical women, there may be no points of difference, when it may be assumed that the two maladies occur together. Hystero-epilepsy presents some remarkable features, especially as regards the condition of tonic rigidity, so that it must always be readily recognized. The influence of pressure on the ovaries and the singular history in these cases will contribute to the facility of diagnosis. Hysterical palsies of every kind are distinguished by the preservation of the electro-contractility, and the occasional absence of electro-sensibility, by the absence of all trophic disturbances, and by the history of hysterical troubles of various kinds. In hysterical hemiplegia there is no facial paralysis, and no apoplectic seizures precede the hemiplegia.

Treatment.—In this malady, above all others, are moral and hygienic measures of most importance. When the hysterical constitution is inherited, prophylactic methods should be pursued from an early period. Self-control should be instilled into the mind from the first dawn of intelligence, and the muscular and digestive systems should be cultivated, while the nervous is trained to subordination. Early hours, substantial food, plain clothing adapted to the needs of the body, should be insisted on, while society, the follies of dress and fashion, and dainties, should be prohibited. The utmost care is necessary in the selection of books for young ladies. The modern novel has done much mischief by cultivating morbid fancies and false notions of the relation of the sexes, etc. Sexual abuses, although less influential than usually supposed to be, do have an injurious effect on the nervous system. If the hysterical condition develops in spite of the precautions advised, remedial measures become necessary. The condition of anæmia must be removed by chalybeates, a generous diet, and suitable exercise. Those tonics are most suitable which have a special direction to the nervous system, as arseniate of iron, strychnine, and the phosphates. As the opposite condition or plethora may exist, although less common than anæmia, iron, arsenic, and strychnine should be avoided, and such remedies as the bromides, gelsemium, and cimicifuga prescribed. For simple hysterical seizures without convulsions, the elixir of valerianate of ammonia, a camphor julep, a little fluid extract of valerian, or a few drops of Hoffman's anodyne, repeated every few minutes, will terminate the seizure. In the convulsive form, as the trismus is difficult to overcome, inhalations of amyl nitrite or of ethyl bromide may be practiced, rectal injections of turpentine, ammoniated valerian, tincture of assafœtida, or, in violent cases, a minute quantity ($\frac{1}{16}$ gr.) of morphine, hypodermatically, may be administered. For the various complications of hysteria the resources of the therapeutist are severely tried. *Migraine* or clavus may be cured by attention to the general health, and by such remedies as guarana, coca, nux vomica, arsenic, aconitine, galvanism, etc. Hysterical aphonia and dysphagia may sometimes be cured instantly by faradic applications. Anæsthesia is most successfully treated by the electric brush, a strong current being applied after drying the part well. The various forms of hysterical paralysis require faradic applications. A single application may overcome paralysis of long standing, especially if the impression made by the electricity is seconded by tact and moral force on the part of the physician. Mitchell has devised a plan of treatment for bed-fast hysterical subjects which seems very successful. It consists in the combined use of massage, faradizations, and forced feeding. Massage consists in friction, kneading and tapping of all the muscles except those of the face, in passive motions of all the joints, and in muscular motions produced by faradic applications. The frictions are made with lard or cacao-butter. The diet consists at first of milk only, but additions are made to it from time to time, until ultimately the feeding is very liberal. No exercise is allowed, but all movements are made for the patient, which is exercise without voluntary effort. Remarkable gain in weight takes place, and when the improvement reaches a certain point systemic voluntary exercise is begun. An important point in Mitchell's treatment is the separation of the patient from all her former associations and the superabundant sympathy of home. She is placed in charge of a nurse, on a diet of milk; hunger takes the place of her indifference to food. She is placed in bed, and not permitted to move; the desire for action grows out of the utterly monotonous idleness. She is acted on by the electrical force, and by the moral force of her new environments, and stimulated to wise thinking by the ingenious suggestions of an acute-minded physician. The result is she is cured.

NEURASTHENIA—SPINAL IRRITATION.

Definition.—The term *neurasthenia* was originally applied by Bouchut [*] to signify the nervous state. This application of the term was revived by Dr. George M. Beard, who also extended its meaning.[†] It is now employed to designate an exhaustion of the nervous system, occurring in a peculiar type of constitution—the neurotic temperament.

Pathogeny.—The most important pathogenetic factor is a peculiar type of nervous system, usually inherited, or constructed by the union of parents having a predominating nervous system. This type is characterized by precocity in the development of the mind; by a nervous system of exceeding susceptibility to impressions of all kinds; by feebleness of the function of digestion; by an imperfect secondary assimilation; and by an inefficient action of the excretory organs. The kind of moral and intellectual training to which such subjects may be submitted — the cultivation of the feelings and emotions, rather than the robuster reasoning faculties—increases the susceptibility and the mobility of the nervous system. These unfortunates acquire the habit of frequent interrogation of their organs, and hence become keenly cognizant of all bodily sensations. They thus lay the groundwork of a selfish valetudinarianism, which needs but little encouragement to expand into any kind of nervous disturbance. These subjects revel in the consciousness of possessing a most irritable nervous system, and dwell in a moral atmosphere of interminable bodily sensations, and are beset by fears that some part may become the seat of pain or other nervous disturbance. Very often a nervous girl has a foolish mother, or some close relative or friend, who is the confidant and repository of her fears, her feelings, and her actual pains, and sympathy, suggestion, and approval, magnify her nervous troubles in every way. The imagination given loose rein, and the attention fixed on some one or all of the organs, the way is prepared, and the nervous state inaugurated. Such is the usual development of neurasthenia in women.

In men, the neurotic state existing, neurasthenia is caused by the various kinds of disturbance to which men are subjected. Self-abuse from puberty to twenty-five; sexual excesses after marriage, or from puberty on; dyspepsia; excessive attention to study or business—are the chief causes of nervous disturbance in men, prepared for it by the training above mentioned. Although to excessive mental work or attention to business is ascribed the cause of nervous derangement, it is probable that the work is only hurtful because carried on under improper conditions.

Various morbid states of the nervous system have been supposed to exist in these cases. The spinal pain and tenderness were at one time considered to indicate congestion, and the back was cupped, blistered, cauterized, and otherwise maltreated. Then the theory of spinal anæmia was propounded by Hammond, and the opposite plan of treatment was carried out. Dr. Jewell, of Chicago, has announced a new theory of the pathogeny of this affection, in which he suggests that the "sensitive tract" of the spinal cord is impaired, and that a "substantial interstitial loss of the ultimate nerve elements" limited to certain "horizontal zones" occurs. These alterations of nutrition he supposes may be due to "under-action" and "over-excitation." As in the present state of knowledge such changes can not be ascertained, this theory will be regarded as probable or not, according to the success with which it reconciles the known facts. As this malady does not shorten life, and as the manifold disturbances of function, connected with it, are intimately associated with a peculiar mental state, we must regard it as a functional disorder, and one chiefly, if not wholly, in the psychic sphere. In fact, there is probably no change in the nerve centers, as no deviation from the normal has been detected on close inspection. Besides the obvious functional derangement of the organs of vegetative life, the most important is in the centers of conscious impressions—in that part of the brain where peripheric excitations of all kinds are translated into consciousness.

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[*] Axenfeld, "Des Névroses," p. 478.

[†] "On Neurasthenia."

Symptoms.—There is no organ or part of the body free from some kind of disturbance. The appetite is poor or capricious, and food occasions distress; in extreme cases, the most bland and simple article excites pain and nausea. Pain is experienced in the left side, in the left hypochondrium, and is often referred to the position of the apex-beat of the heart. Gaseous distention of the stomach and gaseous eructations, sometimes of enormous volume, and consisting of air and carbonic acid, are usual. Torpor of the bowels, the fæces in globular balls, often coated with mucus, sometimes gray and pasty and soft, and flatulence with colic-pains, are always present. The nutrition is usually poor, the subcutaneous fat scanty, the muscles flabby and deficient in power. The pulse is quick, the tension of the vessels low, the heart irritable, and attacks of palpitation are frequent. The vaso-motor system is in a highly mobile state, shown by the chilliness, coldness, and paleness of the hands and feet, which are apt to be covered with a clammy moisture, alternate flushings and pallor of the face, and the sudden and great variations in the tension of the arterial system.

The special senses are variously affected. The eyes are usually very intolerant of light. At first some difficulty in reading is experienced, the page is blurred, and frontal headache follows the attempt to read for even a few minutes. Then the eyes are shaded by turning the head from the light, the blinds are drawn, and reading by artificial light is given up. Soon all use of the eyes is abandoned, the room is kept dark, and the faintest ray of light awakens acute pain and headache, with vertigo. Hearing is apt to be abnormally acute. Harsh sounds occasion distress, and sudden loud noises crash through the brain, producing great pain and giddiness, even faintness ; and thus, gradually, all light and all sound are excluded from the apartment of the most pronounced examples of neurasthenia. Sharp tastes offend in food, and disagreeable odors excite nausea and faintness.

Probably the earliest manifestation of nervous exhaustion, and often the only important one, in men, is an irritability or weakness of the mental faculties. After some exhausting effort, literary, scientific, or in business pursuits, or coming on gradually in the course of the daily occupations, it is found that any sustained attention or thought excites headache, giddiness, or a strong sense of weariness, an obstinate wakefulness that even powerful soporifics only temporarily relieve, accompanied by a feeling of congestion, of vacuity, of coldness, tingling, and creeping sensations in the scalp, sudden concussions, located apparently in the depth of the brain, and various uncomfortable and odd sensations. There is usually a predominant idea connected with the mental state ; there is constant dread of apoplexy, epilepsy, or gradual mental failure. These subjects are liable to paroxysms of headache, frontal or occipital, accompanied by nausea and palpitation of the heart, coldness of the surface, and a tormenting dread of immediate dissolution. They become morally cowardly, weep on the slightest provocation, and have all of the fancies of the hypochondriacal and hysterical.

In the female subjects of this condition, especially, there is much spinal tenderness, with the usual phenomena of *spinal irritation*. Males, also, have tender spinal apophyses, but women suffer more from this condition. The slightest pressure on one of these spots will make them wince, cry out, indeed ; all pressure of stays and of skirts is taken of, and the muscles assume odd positions to relieve the parts of the weight. Lateral curvature is thus induced ; the muscles on one side will be found prominent, rigid, responding with abnormal readiness to faradic excitation, while the corresponding and symmetrical muscles are thin, flattened, and weak. Pains, weakness, and strange sensations are referred to the lower limbs. At the outset walking soon induces fatigue, and is followed by muscular pains and back-ache. Gradually the efforts to take exercise are abandoned, the patient remains in-doors, then does not leave her room, occupying most of the time a rocking-chair and the sofa. The room is jealously closed against light and sound, and the patient reclines in the loosest of clothes on her sofa or in the bed, every organ in a state of rebellion. Finally, all power is lost in the legs ; a strong faradic current causes active contractions of the muscles, but there may be complete anæsthesia.

The sexual functions are always disturbed. In the male there is a decline in the vigor of the erections, and not unfrequently functional impotence results. This topic becomes the predominant idea, and the individual is haunted with a thousand fancies, of which "a loss of manhood" is always the end of the series. In women, the menstrual function is perverted. Sometimes there is amenorrhœa; sometimes menorrhagia. Some disease of the uterus is often the predominant idea with the female subjects of neurasthenia.

Course, Duration, and Termination.—The author's view of the development and course of neurasthenia is as follows: A man, originally possessed of the neurotic type of constitution, has his nervous system rendered additionally impressible by the influences surrounding him. Under the circumstances of this increased excitability of the nervous matter, its elements respond with abnormal readiness to the action of all stimuli, just as a motor or sensory nerve when in an irritable state is functionally readily responsive to stimulation. In a great preponderance of cases the initial disturbance is in the digestive organs, and thence reflex influences proceed to all parts of the nervous system. Sensations which in a normal mind would excite little attention, are in the case of these nervous subjects exaggerated into serious disturbances. The mental condition, after the gastro-intestinal disturbance, is the chief element in the morbid complexus. In no part of the complex of symptoms is the influence of the hysterical or hypochondriacal condition more conspicuous than in the sexual state. The functional impotence, which is a constant symptom, arises for the most part out of the morbid fear of failure. The hebetude of mind and inaptitude for all kinds of mental work are largely dependent on the same morbid apprehension. The various cerebral symptoms which belong to dyspepsia awake in them the acutest apprehension of mental disease. The notion of such disease once started in the mind, every reflex sensation assumes the gravest proportions, and memory, and the power of attention, are apparently much impaired, because the mind, absorbed in the study of the interminable bodily sensations, takes no cognizance of other impressions. Thus, gradually, in one of these nervous subjects, is the condition called neurasthenia evolved, from, it may be, the simplest kind of peripheric irritation, until no function remains undisturbed. In women a similar course of evolution is observed. If we substitute for the worries of business and pecuniary cares, the follies of fashion and the interminable annoyances of housekeeping, we find that women of the neurotic type undergo the same kind of disturbances as men, and these result in them in a similar morbidity. Of course, the peculiarities of their sexual relations determine the direction taken by the morbid phenomena. In what mode it has developed, or whatever form the vagaries have assumed, the course of these cases is protracted. The condition of functional depression of the nervous system recognized, these patients are subjected to the usual routine of tonics, stimulants, and nutrients. They are plied with iron, strychnine, good food, and outdoor air and exercise is enjoined, but to little purpose. Shut up in darkened rooms, with some slave of mother or sister, these wretched invalids pursue their monotonous lives, content, in the midst of their fancies and their sufferings, with those attentions which feed their selfish invalidism. The male neurasthenic, hypochondriacal and yet hopeful of relief, abandons himself to the indulgence of his woes or occupies himself in the fruitless search for a cure which will accord with his own notions.

Treatment.—Moral management and hygienic influences are very important. So long as the female neurasthenic is kept at home, the object of the solicitous attentions and suggestions of some slave of a mother or sister, little can be accomplished. Removed from the home influences, and subjected to an entirely new set of impressions, the first steps toward cure can then be taken. No real change in the mental state of these subjects can be effected until the nutrition is put on a satisfactory footing. Systematic feeding is first to be attempted. One of two methods may be pursued. If hyperæsthesia of the stomach is a pronounced feature, it is necessary to begin with a small amount of aliment at short intervals. It is under these circumstances that the milk-cure proves so valuable. If, however, the stomach permits the reception of suitable aliment, it is only necessary to carry out a proper system of diet, regulated by the nature of the digestive derangements. Next in importance to a proper dietetic management is systematized exercise. It is almost certain that the best mode of resting the brain and mind is to divert the surplus blood into the muscular system. Walking is the best form of exercise, and this must be systematically carried out until the patient thus occupies a large part of his waking movements. It is impossible to exaggerate the value of this expedient in ordinary cases of neurasthenia. Mitchell's plan of treatment, referred to under the preceding article, is well adapted to the cure of these cases. It is probable, however, that a considerable proportion relapse when returned to the influence of their former surroundings.

General faradization, central galvanization, the electric bath, and sparks drawn from various parts, are of great value. The electrical treatment may be advantageously combined with massage, frictions of the whole surface with some animal fat, and regulated exercise. The methods of hydrotherapy, especially residence in some judiciously managed establishment in a mountainous region, is to be commended. Of the medicinal management, cod-liver oil and iron of the nutrient class and strychnine and picrotoxine of the spinal stimulant class may be advised. Arsenic and nux vomica are valuable as special remedies for feeble digestion. Phosphites and phosphates as nerve tonics are useful. It should not be forgotten that medicinal is strictly subordinated to the moral and hygienic management.

CATALEPSY.

Definition.—*Catalepsy* is applied to a state with or without loss of consciousness, in which the cerebral functions are in a state of suspension, and the voluntary muscular system in a position of fixed rigidity.

Pathogeny and Symptoms.—Catalepsy rarely occurs as an independent affection, and is usually associated with certain kinds of mental disorder—with ecstasy, hysteria, and somnambulism. Young, impressionable, and nervous subjects are particularly liable to it. The attacks occur suddenly, and are not indicated beforehand by striking phenomena. It is true that prodromes may occur ; there may be changes in the feelings—sadness, unexpected gayety, a state of apprehension—or actual pain, headache, and general muscular soreness may be felt, or vertigo, yawning, gaping, a condition of unrest, may come on ; but these sensations are neither necessary nor constant. The patient is attacked, in what position soever at the time, as if petrified, but there is no muscular relaxation ; on the contrary, there is a state of tonic rigidity, the antagonistic muscular groups acting with equal tension. The consciousness is abolished in the sense that all exterior objects have vanished, and, although impressions may be received, they produce no reactions. While the mind is in abeyance, the muscular system is in a condition of tonic spasm, resisting passive motion and over which no voluntary control is exerted, and the muscles are suddenly fixed in the position in which they were when the seizure came on, as if set in stone. Although the muscles are not acted on by the will, they afterward submit to passive motion, and remain in any position in which they are placed. But little resistance is then opposed to passive motion ; the members are perfectly flexible, and yet when fixed in a certain position remain immobile, and without trembling or vibrating. The limbs may be put into the most odd and uncomfortable attitudes, and maintain them against gravity for some time, but the muscles at length begin to tremble and ultimately yield according to gravity. The appearance of the patient is very peculiar, sitting or standing immobile in a fixed attitude, staring straight forward and upward, the countenance pale and rigid, breathing scarcely perceptible, the pulse small and weak. On touching the conjunctiva, there are faint movements of the eyelids ; and, if articles of food are placed well back into the pharynx, swallowing is induced, but the organic like the voluntary movements are performed imperfectly. There may be entire abolition of the sensation of touch, of pain, and of reflex movements ; but in other cases the patients have a partial knowledge of events transpiring during the seizures, and in a few instances hyperæsthesia has been noticed. During the attack, the surface is cold, and the temperature falls. When the paroxysm ends, the patient suddenly rouses, takes a deep, sighing inspiration, yawns widely, and gapes loudly, as if waking from a profound and protracted sleep.

Course, Duration, and Termination.—The attacks of catalepsy vary in frequency and severity. They may last a few minutes, several hours, or for days. There is no regularity in the appearance of the attacks, and in the interval the patient may have good health, but usually suffers from hysteria. After the first attacks, the patient may at once resume her ordinary occupation, but repeated recurrences set up a pathological condition of the nervous system, exhibited in the various phenomena of neurasthenia. As catalepsy is associated with certain forms of mental derangement, it is probable that its appearance may sometimes indicate the occurrence of such mental disorder.

Treatment.—Only the protracted cases require attention during the paroxysm. Those cases which continue for days require alimentation by forced measures. The food may be placed well back into the pharynx, or liquids may be introduced through an œsophageal tube passed by the nares. A few minims of amyl nitrite inhaled may suffice to stop the paroxysm, and the hypodermatic injection of morphine may be equally as effective. The usual antispasmodics—as asafœtida, valerian, camphor, turpentine—may be employed by the stomach or rectum. The most important measures are the prophylactic, to prevent the return of the seizures by improving the tone of the nervous system. In anæmic cases, iron, the phosphates, and quinine, are the most appropriate remedies. Change of scene, agreeable variety, occupation affording the mind entertaining employment, are very conducive to the mental and moral stamina of such subjects. Electricity may be employed for the double purpose of arousing patients from the cataleptic state and for improving the tone of the nervous system. The methods of treatment applicable in hysteria are also useful in catalepsy.

PARALYSIS AGITANS.

Definition.—*Paralysis agitans*, or *shaking palsy*, is muscular tremor occurring with loss of power, the subject of the disease being advanced in life.

Causes.—Although rarely seen under forty years of age, it does occur earlier, Duchenne having met with a well-marked example in a man of twenty. The two sexes are about equally affected. Heredity is apparently not concerned in its propagation. The principal causes, besides, probably, a peculiar state of the nervous system, are strong emotion, fright, grief, anxiety and similar moral impressions. Exposure to cold and dampness for a lengthened period, injury to peripheral nerves of an irritative kind, are supposed to cause the disease sometimes. It is said to be more frequent in the Anglo-Saxon race (Charcot).

Pathological Anatomy.—In a certain proportion of cases, not definite, however, no lesions of any kind have been discovered on *post-mortem* examination. In other cases, induration (sclerosis) of the pons, medulla, tubercula quadrigemina, and lateral columns of the cervical cord, has been discovered, but Charcot, with justice, doubts the relation of the lesions to the symptoms. In a third group, the lesions of disseminated sclerosis have been confounded with those of paralysis agitans. A consideration of these facts renders it evident that this disease is a neurosis, a functional disorder.

Symptoms.—In the largest number of cases, paralysis agitans comes on slowly, a slight jerking occurring in a thumb, hand, or foot—in flexion of the thumb and finger, pronation and supination of the forearm. Any effort of the will, as grasping, writing, or walking, will stop the irregular motions. The trembling follows a certain order in its progress from the point of beginning. If, for example, the right hand is first attacked by trembling, after some months or years, the right foot will become affected, then the left hand, next the left foot. Rarely is the middle line crossed, but sometimes this occurs: the right hand first attacked, the next is the left foot. The tremors are often confined to one side of the body for a long time—hemiplegic type; less frequently to both lower extremities—paraplegic type. In some exceptional cases, a feeling of fatigue, or neuralgic pains, precede for some time the trembling, and are experienced in the same limb, which is subsequently attacked by tremors. Sometimes the disease sets in abruptly, in consequence of some sudden shock, and may then affect one member or attack them all simultaneously. In what way soever the disease began, the symptoms of this initial period continue from one to three years, and then pass into the period of fixed intensity. When complete in its development, all the members invaded, the trembling becomes almost incessant, but it is not equally severe at all times. Mental emotion and exercise increase the trembling, and there are periods of exacerbation without any apparent reason, and sleep and chloroform narcosis suspend it. The trembling consists in successive jerks—muscular contraction and relaxation; and in the hand sometimes the thumb and fingers assume a position and movement like the rolling of a pill-mass. The head and neck are not affected, as a rule, but there are exceptions. The muscles of the face are motionless, the countenance fixed and stolid, the muscles of the jaws are unaffected, and there is no nystagmus or oscillations of the eyes. The tongue is somewhat trembling, the lips are compressed, and speech is slow, deliberate, and jerky, as if the pronunciation of each word required a great effort. The muscles of the hand and of the neck, body and extremities, assume a position of characteristic rigidity, preceded by pains and cramp, usually supposed to be rheumatic. The flexors are first and most severely affected. The patient assumes a characteristic attitude, the body bent forward, the neck rigid, making the vertebra prominens still more prominent, the hands flexed and deformed, especially in the fingers, and the whole presenting a strong similarity to the joint troubles of chronic rheumatism. Similar deformations occur in the lower extremities. It occasionally happens that rigidity and deformity occur with the first appearance of the trembling. Notwithstanding the trembling, the motor acts can be performed; they are retarded rather than feeble (Charcot). The muscles are easily tired and the least effort causes a strong sense of fatigue. As a result of the peculiar disability of the muscles, the subjects of paralysis agitans have a peculiar gait. They rise slowly and are deliberate in starting, but, when under way, they go in a dog-trot with the head and body directed forward. Sometimes retropulsion occurs. Given a little jerk backward, they run backward until they fall. Besides the feeling of fatigue just mentioned, these patients suffer from a variety of evil sensations. One of the most distressing is the "fidgets," a feeling of unrest in the limbs associated with the impression of an irresistible necessity for movement. Sensations of pain, touch, and temperature are normal, but a subjective sensation of heat is often felt (Charcot).

Course, Duration, and Termination.—This is a disease of very long duration—it may be thirty years. The first or formative stage lasts from one to three or four years; the period of maximum intensity continues from two or three to twenty years. During this long time there is a progressive increase in the symptoms, until finally the patients are quite disabled, confined to the chair or to the couch. The muscles undergo more or less fatty change, and waste a good deal. At the terminal period very considerable prostration comes on, the urine and fæces are passed involuntarily, and the mind becomes cloudy and wanders. Just before death the trembling may cease entirely.

Diagnosis.—Paralysis agitans and disseminated sclerosis were confounded together, until Charcot pointed out the difference between them, showing that the tremors of the former are always present, while in the latter they occur only when purposive movements are undertaken. In senile trembling the head is chiefly affected, and the movement is merely that of trembling without the peculiar jerking of paralysis agitans; in the former there are not paresis of the muscles, stiffness, deformity of the extremities, and the impulse to forward propulsion and to retropulsion, characteristic of the latter. Mercurial tremor occurs in those who are engaged in some occupation requiring exposure to the vapor of mercury, and it differs from paralysis agitans in being purposive, accompanied by troubles of coördination, defects of vision, by a grayish-blue line along the margin of the gums, by a fetid breath, and sometimes ptyalism.

Treatment.—There are several remedies that moderate the trembling—hyoscyamine, according to Charcot, is the best, and in the author's observation no remedy has acted so efficiently as this. Gelsemium sometimes is quite beneficial, but it must be given in quantity that will make an impression. To lessen the retrograde changes, the best results are obtained from the chloride of gold and sodium, with or without corrosive sublimate in small doses, given persistently, oxide or nitrate of silver and the lactophosphate of lime with arsenic, continued steadily for months at a time. Eulenberg has had good results from the hypodermatic injection of arsenic, and Ogle from extract of physostigma. Monobromide of camphor has appeared useful in some cases. The milder applications of hydrotherapy have done good in a few instances. From the variety and diversity of the remedies recommended, it is apparent that no plan of treatment has been satisfactory. There is a general agreement that the galvanic current is useless. Eulenberg* says he has seen no good results from it; Erb's and Rosenthal's experience is the same.

CHOREA.

Definition.—By *chorea* is meant a functional nervous disorder, characterized by defects of voluntary coördination, and by irregular spasmodic movements in certain groups of muscles.

Causes.—A peculiar mobility and impressionability of the centers of coördination are, doubtless, transmitted by inheritance. The mode of life, education, and training may induce this unnatural mobility. The disease usually makes its appearance about the second dentition, or at the period of puberty. When the predisposition exists, various causes may excite the morbid complexus. Among the most important of the causative influences is rheumatism, or rheumatic endo- and exocarditis. The closeness of the relation is variously stated. Professor Sée is at one extreme, for he finds in one hundred and twenty-eight cases of chorea sixty-four cases of acute rheumatism. Steiner, of Prague, is at the other extreme, for, in a series of two hundred and fifty cases of chorea, only four resulted from acute rheumatism. It is impossible to harmonize these observations. According to the author's experience, the proportion of rheumatism to chorea is about one to eight—much more than Steiner's, and less than Sée's. Intestinal worms, sexual abuses, amenorrhœa, anæmia, and strong moral emotions, are frequent exciting causes, and to these must be added pregnancy.

Pathological Anatomy.—There are no constant changes in the anatomical elements. As a large proportion of cases recover, it is probable that the derangements are functional. As so many cases are complicated by endocardial alterations, embolic obstruction of the minute vessels of the corpus striatum, or optic thalamus, has been proposed to account for the morbid phenomena. Hughlings Jackson has especially supported this view. As emboli have been discovered in some cases, it seems probable that this explanation is occasionally true. But various changes have been discovered: thus Meynert found changes in the cerebral cortex, and Elischer has recently detected nuclear proliferation, thickening of the adventitia of the minute vessels, and hyperplasia of the neuroglia in the corpus striatum.* Localized softenings in various parts of the cerebro-spinal axis have been noticed, but no relation can be traced between such softenings and chorea, except those situated in the corpus striatum.

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* "Lehrbuch der funktionellen Nervenkrankheiten," *op. cit.*, p. 711.

Symptoms.—A sudden terror has produced a fully developed chorea in an extremely nervous child, but usually the onset of the disease is gradual. At first the child appears to have adopted some trick or a grimace, or an ugly motion of the shoulder or arm. Then irregular jactitations become more common in the face and upper extremities. The choreic movements may be limited to one side of the body, when it is known as hemi-chorea, or to the upper or lower extremities. In a severe case all of the voluntary muscles of the body are engaged in choreic movements; the muscles of the face are distorted into endless grimaces; the eyes roll (nystagmus), and, the muscles acting unequally, there is strabismus; the tongue is jerked about the mouth, so that speech is difficult or unintelligible, and is sucked into the throat with an audible smack; the arms are troubled by endless jactitations, the fingers are twisted into all conceivable shapes, and writing, using the knife and fork, and holding any object are impossible; walking is irregular, the legs catch each other or trip over objects; breathing is spasmodic and sighing; the heart's action is tumultuous, irregular, and apparently also choreic; a soft-blowing murmur may be audible at the base, or a loud, churning systolic murmur, heard with greatest intensity in the mitral area. In the severest cases the patient can not remain in any position, but all the voluntary muscles are simultaneously engaged in the most violent and disorderly movements. The features are swollen and bloated; blood is seen about the teeth; the extremities are bruised and bleeding by the continual knocking of bony prominences against the wall, the bed, or the floor. In the mild cases the jactitations are occasional and not severe, and cease during the night, permitting quiet repose. In the severe cases only snatches of sleep are obtained, the jerking of the muscles coming on after very short periods of quiet. In the severest cases the jactitations are incessant, and sleep is impossible. In all cases of chorea sleep is apt to be disturbed by unpleasant dreams, and somnambulism is by no means uncommon. There is general exaltation of the senses of touch and pain, and the reflexes are increased. Tenderness of the spine, especially of the cervical and upper dorsal regions, is a constant symptom. Weakness or perversion of mind is observed in all decided cases, but usually impaired memory, stupidity, irritability, and morbid impulses have been observed.

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* "Ueber die Veränderungen in den peripheren Nerven und in Rückenmark bei Chorea Minor," Virchow's "Archiv," lxi, p. 485.

Course, Duration, and Termination.—The course of chorea is chronic and continuous, and the duration of ordinary cases is from one to three months. Although regarded as self-limited and tending to spontaneous recovery in two or three weeks by some authorities, it is really much influenced as to its course and duration by appropriate treatment. It may continue for a number of months, for years in fact, but this is excessively rare. Exacerbations and relapses are very common. Those having attacks at about seven years of age are apt to experience seizures up to puberty. If occurring in the first pregnancy, it is apt to occur in subsequent pregnancies. The most intractable cases, according to the author's experience, have been those of the first pregnancy. Although the termination is usually in health, death may result from the exhaustion due to the incessant jactitations, want of food, and loss of sleep. The existence of pregnancy is a serious complication, for, besides the danger of miscarriage, the severity of the disease induces rapid exhaustion. Jaccoud collected thirty-one cases of the chorea of pregnancy, and of these four died. After delivery the convulsions cease, but very rarely before delivery.

Diagnosis.—Chorea is accompanied by such pronounced symptoms that it can hardly be mistaken for any other disease as it occurs in children. It may be confounded with disseminated sclerosis which appears in young subjects, and which has for a prominent symptom muscular tremor, but the tremors are perceived only on intentional movements, and cease when the muscles are at rest. Furthermore, this disease is accompanied by pareses of the muscles and the rigidity of extension, and often sets in with an apoplectic attack and other formidable symptoms; and its course and behavior are so different in all other respects from the tremor, that the least attention ought to prevent error. Paralysis agitans differs from chorea in the age of the subject, the deformity of the hands, the muscular rigidity, the shape assumed by the spine, and the character of the gait, and in the subsequent course and termination.

Treatment.—Excellent results have been obtained by a simple hygienic treatment—by confinement to bed in a darkened and quiet room, and careful but generous alimentation. As moral causes, excitement and bad hygiene, are very influential in causing the disease, supplying the patients with the opposite conditions ought to effect improvement. Treated in this way, it was ascertained at Guy's Hospital that chorea has a tendency to spontaneous cure in two or three weeks

It is important to give to choreic subjects sound sleep—to suspend the jactitations during ten hours. This is best accomplished by the combined use of morphine and chloral. A generous diet should be directed, and the utmost quiet and repose enjoined. Any eccentric irritation, as worms in the intestines, impacted fæces, elongated prepuce, or sexual excess, should be corrected. Anæmia requires the free administration of iron, lactophosphate of lime, and strychnine. The remedies to stop the choreic movements consist of the mineral tonic group—arsenic, the zinc preparations, ammoniated copper, and iron ; of the vegetable paralysant group, as succus conii, gelsemium, physostigma ; and the anodyne group, as opium, chloroform, chloral, bromide of potassium. Of the mineral tonic remedies the best results are obtained from arsenic, of which very large doses are easily borne. In some obstinate cases the hypodermatic injection of arsenic has achieved successes. In the most violent cases, chloroform may be indispensable to give even a few minutes' repose. In these violent cases, enormous, almost incredible doses of morphine were given by Trousseau with advantage. Mild cases are benefited by ether-spray directed against the spine for a few minutes every day. Galvanization is also serviceable. A stabile current, not too strong, should be applied to the spine and to the principal bundles of spinal nerves. Hydrotherapy, in the form of a wet pack, and douche to the spine, has been useful in many cases.

WRITER'S CRAMP.

Definition.—*Writer's cramp* is a faulty term, but no really better designation has been proposed. It is intended to express the idea of a muscular disability produced by overuse in a strained position of certain muscles. It is called writer's cramp because so many cases have arisen from this employment. The same disability occurs to pianists, to seamstresses, and some other employments requiring the continuous use of the same group of muscles.

Pathogeny and Symptoms.—There is not an actual condition of cramp ; the affected muscles are not paralyzed, and are equal to all other work, except the particular duty in which they acquired the disability. Duchenne well expresses it when he says there is an impotence in respect to the particular position and movements involved in writing. There is no disorder of intelligence, no lack of ideas, and the motorial apparatus is intact, but the muscles, so long and constantly employed in the prehension of the pen, the poising of the hand and forearm, and in the movement of the pen (Poore *), become unequal to the task. The growth of the disability is slow. Fatigue in the much used muscles, pain in the forearm, in the wrist, and in the hand, are experienced. So strong is the sense of fatigue, and it may be pain in the arm, that rest is often taken; the arm is steadied, and the pen is seized with a firmer grip. Other muscles are called into action, and great efforts are made to relieve the fatigued muscles by writing with the whole arm. The writing changes its character and becomes irregular; the muscles of the first three fingers, after a time, are given to fibrillary trembling. Finally writing becomes impossible; the pen is taken up, a strong effort of the will tries to force the muscles to the task, but they obstinately refuse to execute the necessary movements. In a perfectly normal state, writing is so constantly and for such a long period carried on, that the supervision of the higher centers over the muscular movements ceases to be exercised: in other words, the act of writing becomes largely automatic. When such a muscular disability occurs, the attention must be again directed to the act, and then a new element of discord is introduced. Besides fibrillary trembling, a condition of tonic spasm seizes the muscles of the thumb and the flexors of the fingers. These involuntary contractions or spasms of the muscles sometimes also involve the extensors, and thus a condition of *ataxia* results. There is still another group of cases in which a marked paresis or weakness of the flexors of the thumb and fingers takes place, and fibrillary trembling frequently coincides with the weakness. This group is called the *paralytic* form. There is still another group in which the flexors and extensors are occupied by cramps, there is no trembling, no sense of fatigue, and the sensibility is intact. In the paralytic group the electro-sensibility and the electro-contractility are reduced; in the spasmodic group, the electro-sensibility and contractility are either exaggerated or normal.

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* "The Practitioner" (London), 1879.

Course, Duration, and Termination.—The course of writer's cramp is very chronic and the duration indefinite. It is more often than is supposed the precursor of more serious ailments of the nervous system. If, with the first symptoms, entire rest be given to the affected member, a cure may be readily effected; but, when the disability is complete, the prognosis as to cure is very gloomy. If it be true, as the author's observation has led him to conclude, that writer's cramp is often followed by other nervous diseases, no case is without importance, and the management should include instructions as to manner of life and regimen, to avoid future complications.

Treatment.—As soon as the symptoms of writer's cramp become manifest, writing should be relinquished immediately, and the muscles be given rest for several months. Rest may remove all the symptoms, and subsequently moderation in the amount of writing and giving sufficient intervals of rest will entirely obviate the tendency to cramp or paresis. Much attention should be given to the position of the fingers, and to the amount of effort necessary. A large pen-holder and an easy, unembarrassed manner of grasping the pen are of much importance. When the case is complete, and writing becomes impossible, a cure is not to be hoped for; but such amelioration may be effected as to permit a very little daily use of the hand in writing. There are two local remedies of real value—galvanism and massage. A current from ten to fifteen of Siemens and Halske's elements should be passed daily for a few minutes through the affected muscles. If spasm and fatigue are the conditions of the muscles, a stabile current is to be preferred; if the muscles are weak, a labile current should be used. The forearm, the muscles of the thumb, and the interossei should be gently rubbed and kneaded for a few minutes previously to the application of electricity. If the general health is depressed, good effects are obtained from strychnine; but this agent does harm if the nervous system is excitable and the circulation active. The phosphates, quinine, and cod-liver oil, should be prescribed if the health is poor.

TETANUS.

Definition.—By *tetanus* is meant a disease characterized by paroxysmal tonic contractions of the voluntary muscles, and due to an exaltation of the reflex function of the spinal cord.

Causes.—Tetanus may be produced by intrinsic or central lesions and extrinsic or peripheral lesions. The latter are more important than the former. As the best example of a tetanic condition due to centric causes may be mentioned the action of those agents which increase the reflex excitability of the spinal cord, namely, strychnia, brucia, and thebaia. The extrinsic causes are wounds and injuries of various kinds, especially those involving nerves, many of which are insignificant—for example, the prick of a needle, the extraction of a tooth, perforating the ears for ear-rings, or bleeding—each of which has caused tetanus. Internal traumatic injuries may produce the same result. Tetanus has followed parturition and uterine diseases; and the so-called idiopathic tetanus has supervened upon inflammatory exudations, involving the pneumogastric or phrenic nerves. The severity of the injury bears no relation to the frequency or violence of the attacks. When a wound is cicatrizing, tetanus is more apt to occur, especially if the cicatrix is so situated as to compress a nerve. The situation of a wound has more influence—those of the extremities having the greatest effect. *Trismus neonatorum*, tetanus of the new-born, occurs usually from the fifth to the twelfth day, and is attributed to section of the funis and a subsequent inflammation. Tetanus also succeeds to circumcision. Much influence is ascribed to cold by some writers. It is probably true that wounded men, exposed to cold, are more liable to the disease. The free use of cold water as a dressing for wounds, during the rebellion, was responsible for many cases, it is supposed by competent judges. On the other hand, tetanus is a common malady in tropical countries.

Pathological Anatomy.—The changes occurring in tetanus are found in various parts of the cord, but chiefly in the medulla oblongata, in the lumbar region, in the gray substance around the central canal, and in the anterior horns. Very considerable dilatation of the vessels is always found. Exudation of a semi-fluid, colloid substance, hyperplasia of the neuroglia, and abundant nuclear proliferation in the gray matter, have been observed in the more recent microscopical investigations.

Symptoms.—The onset of the disease varies according to the cause. When due to a wound, there are changes in its character as the disease is about to develop: the cicatrization ceases, the suppuration presents a different aspect, the wound becomes irritable, tender, and red, and pains shoot along toward the body. When caused by cold, there is chilliness, followed by fever, and stiffness of the neck is felt. The first manifestation of the tetanic paroxysm is, in a great majority of cases, in the motor branches of the fifth, which innervate the masseters and internal pterygoids, and the jaws are set in a condition of rigidity. To this tetanic fixation of the jaw is applied the term *trismus.* The attempt to swallow excites cramp of the pharynx, and is therefore difficult and painful. Next, the post-cervical muscles become rigid, and the head is held back. The muscles of the face now take a fixed position, the lips are retracted, exposing the teeth; the brow is corrugated, giving to the countenance a mixed expression of anguish and laughter—the *risus sardonicus.* The muscular rigidity now extends to the trunk and extremities, and hence the whole body, while helpless, is immovable and rigid. As the spinal muscles are more tense and more powerfully acted on, the body is bent, and may rest only on the occiput and heels. This position is entitled *opisthotonos.* Less frequently, the body is bent in the opposite direction, or forward—a position known as *emprosthotonos.* Still more rarely the inclination is lateral, or *pleurosthotonos.* The condition of excitation is not the same all along the spinal canal, for we find that the flexors of the upper and the extensors of the lower extremities are comparatively more active. In the beginning of the attack, the rigidity is not constant, does not affect all the muscles equally, and may pass from one to another group. There are remissions also at first, during which there may be complete relaxation. But the paroxysms become more frequent and severe, and are presently excited by the slightest movement. So exquisitely excitable is the reflex faculty, that the least possible peripheral impression brings on a spasm—a mere touch, a current of air, the reflection from a mirror or surface of water, will excite it. At the moment of the spasm a sudden tonic contraction seizes all of the voluntary muscles, the face is horribly distorted, the spine is bent, the body resting on the head and heels, the abdomen retracted, respiration suspended, the feet incurved and extended, the hands violently clinched and drawn in with the forearms toward the body. During the convulsion a severe pain is felt at the epigastrium, and extends through to the back. The muscles so violently acted on are very painful, and even rupture of fibers, sometimes of a muscle, may take place. The paroxysm soon reaches its maximum and then subsides, and during the interval between them the patient breathes more easily, and is able to swallow a little. The frequency with which the paroxysms come on, their violence and duration, furnish the measure of the importance of the case. Some sleep may be obtained in the interval between the paroxysms, but on awaking the attacks of spasm are resumed, and in severe cases sleep is entirely prevented. Meanwhile, the mental powers are unimpaired, and sensibility and the special senses remain normal. In a few instances diminution of sensibility has been noted. As muscular activity is a great source of animal heat, it is not surprising that in this disease there should be rise of temperature. The fever does not pursue any special type, but at death it may attain to 104° or 105° Fahr., and rise even higher for an hour or two after death. Profuse sweats also occur. Respiration during the spasms is carried on by the diaphragm only, and the pulse becomes hard and very rapid. The voice is harsh, guttural, and sometimes speech is unintelligible. The mouth is dry, the saliva viscid, deglutition almost impossible, and constipation is the rule. The urine is normal, or scanty, usually alkaline, and sometimes contains sugar.

Course, Duration, and Termination.—The course of tetanus may be very acute, or more protracted, when it is known as chronic tetanus. In the acute form an early termination is caused by tetanic fixation of the muscles of respiration. In the chronic form the intervals between the paroxysms are longer ; the patient has an opportunity to obtain some sleep and to take food. In the tetanus of the new-born, and in toxic tetanus, the duration is shorter than in the traumatic, the paroxysms succeed each other rapidly, and death occurs in asphyxia. Idiopathic tetanus is not so violent, as a rule, and the prognosis is hence more favorable. Traumatic tetanus is always serious, but the case may be regarded as more favorable when the intervals between the paroxysms are long enough to permit sleep and alimentation, and the paroxysms are less dangerous to respiration. The case is still more favorable if, after the second day, there is no increase in the number and severity of the paroxysms.

Diagnosis.—Tetanus is distinguished from strychnine-poisoning by the sudden onset and quick termination of the latter, and by the presence of a wound or some other cause of the seizure. In spinal meningitis there are tonic spasms of the muscles, but the rigidity is not paroxysmal, and there are no intervals of entire cessation of the morbid action; there is not the great reflex excitability of tetanus and the occurrence of cramps on slight irritation peculiar to that disease, and in spinal meningitis the tonic rigidity is succeeded by paralysis. Hydrophobia is very similar to tetanus, but it develops more slowly; there is a special antipathy to water and inability to take it when other articles may be swallowed, and a peculiar hawking noise is made, to dislodge a little viscid secretion from the throat, peculiar to this disease. Trismus may be limited to the muscles of mastication, and may be produced by colds and exposure, but it is confined to these muscles and does not become generalized. Those cases occurring in the course of cerebral disease are also diagnosticated by the symptoms of such diseases, which have no relation to tetanus.

Treatment.—Whenever an obvious cause exists it must be removed. If a wound, splinters of bone and foreign bodies should be searched for; if a cicatrix, it should be dissected out; if an injured nerve, it should be divided. The remedies which have been most successful are those which diminish the reflex function of the spinal cord. Bromide of potassium seems to have been the most successful agent thus far employed. It must be given in very large doses—from one to two drachms every four hours, until the spasms are decidedly diminished, when the quantity may be somewhat reduced. Given early, and the effect maintained until the spasms cease, it must be regarded as the best remedy in view of the large proportion of cures. Next to the bromide is curara, which acts on the end-organs of the nerves and on the reflex faculty. This must be given hypodermatically, and the effect produced must be the guide. As curara is a very uncertain substance in its composition, the dose necessary can only be determined by trial, but, inasmuch as one eighth of a grain has been administered at a dose, it will be prudent to commence with one fortieth of a grain, and increase it until some effect on the spasms has been caused. Nicotine has similar properties and powers, and has been used hypodermatically in tetanus and in strychnine-poisoning with success. The author has seen a very severe case of traumatic tetanus treated successfully with the wine of tobacco. Physostigma and eserine have been now employed in a large number of cases and with excellent results. Eserine can be given subcutaneously, beginning at one sixtieth of a grain and increasing it until some effect is produced on the spasms. Cannabis Indica has also arrested some cases of tetanus, and is a very promising remedy. Recently urethane has been proved to have the most complete antagonism to strychnine, and is strongly recommended as a remedy for tetanus. The spinal ice-bag and the continuous current have proved palliative. Warm baths and the vapor-bath have given comfort, and have exerted a temporary influence over the spasms. An estimate of the value of a remedy is much affected by the period at which it is administered, for the longer the case has lasted the more hopeful. The nutrition of cases of tetanus is highly important, and from the beginning they should be carefully fed. Noises and excitement, every form of peripheric irritation, and emotion of all kinds, should be excluded. As there is strong temptation to use ether and chloroform freely because of the relief they afford, the author desires to caution his readers, because of the injury so often done by them.

DISEASES OF THE PERIPHERAL NERVES.

NEURITIS.

Definition.—The word *neuritis* signifies inflammation of a nerve, but there are several distinctive maladies which may be grouped under this designation. There is a simple neuritis, in which one or more nerves may be affected by some local cause, usually trauma, and this may be either an *acute* or *chronic* inflammation. *Toxic neuritis* is that form of the disease induced by some poison introduced from without, as lead, copper, arsenic, etc. *Diathetic neuritis* arises from some systemic condition, such as rheumatism, gout, syphilis, etc., or it is due to the blood changes in typhoid, diphtheria, scarlet fever, and similar diseases. Again, neuritis, in consequence of conditions not well understood, manifests a tendency to spread from its point of origin and involve many other nerve trunks, whence we have *ascending neuritis*, *multiple neuritis*, and *progressive multiple neuritis*.

Causes.—The influences affecting the inflamed nerves are necessarily various. Simple neuritis is produced by wounds, injuries, and by the transference of the morbid action from a neighboring inflamed tissue. Thus, intercostal neuritis is caused by an adjacent pleuritis, or tuberculosis of the lung ; sciatica, by a pelvic abscess or inflamed hæmorrhoids ; and caries of bone may involve a nerve or plexus of nerves in the vicinity. As neuritis may occur in various nerve trunks simultaneously, and without apparent cause, there is, probably, a peculiar type of nervous system in which such an action is prone to take place. It is probable, also, that the occurrence of neuritis, in cases of fever and septic diseases, is due to the constitutional type of the affected individual, for such an accident is quite unusual. The special causes which originate and maintain progressive multiple neuritis are quite unknown.

Pathological Anatomy.—The first step in the process is hyperæmia : exudation takes place into the nerve, which becomes softened and ultimately breaks down into a diffluent mass. Migration of white corpuscles takes place into the neurilemma, an exudation partly serous, partly fibrinous, and minute extravasations occur between the fasciculi, and then suppuration and softening result. Recovery may ensue before disintegration of the nerve elements is produced. The fibrinous exudation undergoes the usual changes—the watery part is absorbed, the solid matters and the corpuscular elements become fatty and are then taken up, and health is restored. In the chronic form of neuritis the change is less toward pus-formation and softening, and more to hyperplasia of the connective tissue. The nerve forms intimate adhesions to the neighboring connective tissue, the medulla undergoes fatty degeneration, and the nerve-fibers and axis-cylinder atrophy. These changes may occur in particular parts of the nerve, giving it a knobbed appearance, whence the term *neuritis nodosa*. It is important to note that when inflammation occurs in a nerve it may extend from the point first diseased upward (*neuritis ascendens*), or downward (*neuritis descendens*). By the extension of an ascending neuritis the spinal cord may be ultimately affected.

Symptoms.—If an important nerve or plexus is inflamed, there may be some fever preceded by chilliness, or a decided chill, headache, and general muscular soreness; but the most pronounced symptom is pain in the nerve, not only at the point inflamed, but spreading thence over the peripheral distribution. The pain is of a very distressing kind; it is a burning, tingling, tearing, and intense pain, and is increased by motion or pressure. There is a high degree of sensitiveness in the region of the inflammation; numbness and formication are mixed with the pain, and ultimately the parts supplied by the nerve become anæsthetic, which means destruction of the nerve, or pressure sufficient to prevent the transmission of impulses. If the nerve inflamed be motor in function as well as sensory, there will occur spasmodic contractions and cramps in the muscles to which the nerve is distributed; then will follow paresis, and ultimately paralysis, if the nerve is compressed or destroyed. Besides the general fever accompanying the neuritis, there is a local elevation of temperature in all the region of distribution of the nerve. In the chronic form there do not occur the constitutional symptoms which are present in the acute form, but pain and other symptoms of sensory irritation, and cramps and other symptoms of motor irritation, do appear. Besides the effects of neuritis within the distribution of the affected nerve, various reflex and radiation phenomena are manifest. Pain is felt in all the branches of the same plexus, and cramp in the muscles innervated from the same source. Wasting and degeneration of the muscles and anæsthesia of the parts innervated by the affected nerve are results of the neuritis. Various trophic disturbances are also caused. Various forms of cutaneous eruptions appear—herpes, eczema, and "glossy skin"; the nails become clubbed, the hair falls out, and the joints swell and change in structure. The affected nerve in the stage of irritation responds more readily to electric currents; if the nerve is simply compressed the muscles may respond normally, yet if destroyed there will be no reaction to faradic stimulation, but to the galvanic : in other words, after the increased excitability to electric stimulation, the characteristic reactions of degeneration will come on.

Progressive Multiple Neuritis.—Simple acute or chronic neuritis manifests a tendency to extend from the point of original mischief upward. In progressive multiple neuritis, this tendency to extension upward is the most distinctive characteristic of the malady. It is also widely diffused, numerous important nerve-trunks becoming simultaneously affected. It is either occurring more frequently than formerly, or it is a comparatively new disease. It may be that more exact knowledge renders its differentiation more certain. Since Dumenil published his observations, the first that had been made, various cases have been reported. After Dumenil, examples of the disease, with comments, were published by Eichhorst,* Jaffroy,† and others. Last year Dr. Webber, of Boston, published an important memoir, based on cases he had observed. Some cases have been encountered by the author, and he has now a very complete example in his charge. It is probable that this disease, sometimes, has been mistaken for progressive muscular atrophy.

Progressive multiple neuritis is an acute affection, marked at the onset by chilliness, fever, and tingling pains, with coldness of the hands and feet. As in all acute inflammatory affections of the nervous system, there are two distinct groups of symptoms : those of the first stage, significant of irritation ; those of the second stage, indicating depression. The irritation symptoms are due to the congestion of the nerve sheaths and trunks, and the beginning exudation ; the anatomical elements as yet remaining intact, respond to the irritation caused by new materials in the tissues. As compound nerves are those affected, obviously the symptoms must include both sensibility and motility, pain and spasm being the result. Tingling, burning, and lightning-like strokes in the paths of distribution of the nerve-trunks of the extremities are experienced. The ulnar nerve and its terminals appear to be specially affected. The nerves, the seat of pain, are found to be very tender on pressure, and the intramuscular filaments being also affected, any muscular movement causes suffering, which may be so severe that the patient maintains as complete repose as possible. During this period of heightened sensibility there occur, also, motor phenomena of a corresponding character. With the onset of the irritation of the motor nerve-fibers, the muscles innervated by them become spastic, tense, and disposed to contract, or may be seized with spasms. The reflexes are heightened, and the responses to stimulation assume the character of tonic cramp. The electrical reactions correspond ; there is an increased readiness of response on the part of the sensory nerves with pain, and of the motor nerves with tetanized contraction. But the morbid action continuing, in a short time the increased sensibility is succeeded by anæsthesia and analgesia. The tactile sense is blunted so that the points of the æsthesiometer are only recognized when widely separated. The senses of pressure, of temperature, and of locality become equally blunted. The muscles grow weak, paretic, and are presently paralyzed, and with the paralysis rapid wasting ensues. The atrophic degeneration begins in the small muscles of the hand, and thence extends to the forearm, and in these parts it continues more pronounced than elsewhere, but all the paralyzed muscles undergo more or less change, and exhibit finally the characteristic reactions of degeneration. The cutaneous and tendon reflexes are also abolished. The skin, nails, and hair undergo atrophic changes.

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* Virchow's "Archiv," vol. lxix. "Neuritis Acuta Progressiva."

† "Arch. de Physiol. Norm. et Path.," 1879, p. 172. "Névrite parenchymateuse, spontanée, généralisée ou partielle."

The acute symptoms last from a few days to a few weeks, and during this period the sensory and motor disturbances become well defined. The disease then assumes a chronic character, and continues on from two months to two years. In the favorable cases, the disease does not diffuse so widely, the paralysis is less complete, and the trophic changes are not so pronounced. In the most favorable cases, there may be permanent wasting and deformity of the muscles innervated by the ulnar nerve, the little and ring finger remaining contracted, shriveled, and almost useless, and the hypothenar eminence shrunken. In the unfavorable cases, the chief nerve-trunks of the extremities are invaded, the nerve-roots and the spinal cord become diseased, and the four members are disabled. An extension of the neuritis may take place to the medulla with the production of the characteristic results in the respiratory and circulatory systems, or the patient may be cut off by an intercurrent malady, as pleuritis, pneumonia, or phthisis.

The prognosis as regards danger to life is favorable, as but few cases die. Protracted suffering, paralyses, and deformity may be looked for in the more chronic cases, and recovery, when it ensues, is apt to be clouded by the disabilities.

Course, Duration, and Termination.—The acute form is necessarily of short duration. Recovery ensues, permanent disability results, or it becomes chronic. Restoration is possible only before disintegration of the nerve. The chronic form has no fixed duration. Recovery is more likely to ensue when there has occurred a simple injury or exterior pressure, which may be removed, than when an idiopathic or rheumatic inflammation has taken place. The latter are apt to become very protracted, to have periods of remission and exacerbation, thus continuing for years. The prognosis will be largely determined by the character of the symptoms—pain and muscular cramps, indicating the stage of irritation—anæsthesia and paralysis, the stage of injury to the nerve-trunk. Very important in this connection is the electrical diagnosis; for, if the irritability of the muscles to the faradic current is preserved, the nerves are still intact, and *vice versa*. As neuritis manifests a strong tendency to ascend, in the course of the malady secondary degeneration of the spinal cord may ultimately take place.*

Diagnosis.—The differentiation of neuritis from myalgia is effected by reference to the points of tenderness—to the symptoms of irritation, succeeded by those of depression of a nerve; from neuralgia, by the fever in the acute form, by the changes in the trophic condition of the skin, and by the state of the muscles and the reactions of the faradic current.

Treatment.—The various causes of the disease must be removed. Here surgical treatment of wounds and injuries may be invaluable. In acute cases of plethoric and vigorous subjects, leeches should be applied along the course of the nerve. A full dose of morphine and quinine should at once be given (gr. ss.—gr. xv for an adult), and the tincture of aconite-root (two drops every two hours); or morphine may be given subcutaneously if the pain is severe. In the chronic cases, the most effective remedies are galvanism and the hypodermatic injection of morphine. The positive pole is placed on the tender spot or spots, and the negative at the peripheral expansion, daily application of a few minutes' duration being made. A succession of flying-blisters, or the electric brush, or the oleate of morphine, may be used locally, the iodide of potassium, colchicum, etc., internally in the more obstinate cases.

ATROPHY OF THE NERVES.

Pathogeny.—Atrophy of the nerves arises from various causes: from central diseases, of which examples are afforded by posterior spinal sclerosis, progressive bulbar paralysis, infantile paralysis, etc.; from peripheric lesions, as injuries by wounds, or compression of tumors, etc.

Symptoms.—The disturbances by atrophy are part of the morbid complexus of various affections, and consist in depression of function, wasting of the muscles, paralysis, and, as regards the sensory nerves, anæsthesia.

NEURALGIA—NEURALGIA OF THE FIFTH NERVE.

Definition.—Neuralgia of the fifth nerve has received various designations—*prosopalgia*, *tic-douloureux*, *Fothergill's disease*, etc.

Causes.—The causes of tic-douloureux may be comprehended in three groups—constitutional, immediate, and remote. Heredity is an important factor, since this disease is one of numerous maladies possible to the neurotic temperament or disposition. It is not unfrequently associated with epilepsy, as Trousseau was the first to point out. It may occur at any age, but is more frequent from the middle period, on, and in women at the climacteric period. Anstie* insists on the importance of the degenerative changes of age as causes of the origin and of the intractable character of some cases. The female sex seem more susceptible than males. Certain dyscrasiæ, as lead, syphilis, malaria, etc., are undoubtedly causative. Anæmia, amenorrhœa, a depressed state of the bodily functions, the exhaustion induced by excesses in venery, gout, and rheumatism, are predisposing causes. Psychical impressions, especially if depressing, are held by Anstie to be causative. Changes in the structure of the nerve, tumors, exostoses, and aneurisms, caries of the bones, periostitis, gummata, etc., are among the immediate causes. Decayed teeth, indigestion, worms, constipation, menstrual derangements, etc., are among the remote causes.

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* Vulpian, "Archives de Physiologie," vol. ii, 1869, p. 221, "Expériences relatives à la pathogénie des atrophies secondaires de la moelle épinière."

Pathological Anatomy.—The changes of neuritis have been sometimes observed in the trunk of the nerve and in the ganglion of Gasser. More frequently no changes have been noted. The nerve is more often affected by exterior pressure. In one of the most severe cases ever witnessed by the author, the nerve was impinged on by an aneurism of the basilar artery, and was very much thickened and softened. Probably the most frequent pathological condition is the pressure of an exostosis, or other form of tumor, on the trunk of the nerve within the cranium.

Symptoms.—The usual history is that of gradually increasing pain in the face or teeth. At first the attacks are regarded as merely toothache, and tooth after tooth is extracted in the vain hope of finding the painful one. It may be months before the pain assumes the characteristic expression. Then distinct paroxysms occur, than which nothing can be more horrible. A sudden pain pierces the face, the muscles of that side are convulsed, the eye is injected, and the tears flow—the patient starts up with a terrible groan, rubs the cheek vigorously, wrings his hands, cries out in the extremity of his agony, rushes about his apartment, and it may be suddenly the pain ceases and the paroxysm is over, or it gradually subsides. At first these attacks may be weeks, even months apart, but after a time they get more numerous. In the interval between the seizures there may be entire freedom from pain, but in many cases there is nearly constant soreness, or aching, in the jaws or eyes. When the pain is wholly paroxysmal, the attacks are more frequent, and, in the interval between them, the patient experiences a tense feeling in the affected region as if the slightest movement on his part would excite a paroxysm. When this sensation comes on, he durst not move, he can not be spoken to, every muscle is in a state of tension and immovable, he hardly breathes, he looks straight before him in an attitude of suspense and apprehension. In spite of the dreadful energy of the self-control, his effort often fails, the pain comes on with a lightning-stroke, his teeth set hard, the face pales, the pupil dilates ; then he abandons himself to his suffering, he starts up with a groan, and repeats the rubbing, the wringing of hands, the cries, etc. Ultimately so sensitive become the peripheral nerves, that the slightest touch, a breath of air, excites the paroxysm, and the attempt to take food produces the most frightful torments, the face is thrown into spasms, tears run down the cheeks, and the patient utters horrible groans. So dreadful is the aspect of this suffering, that these unfortunates must needs eat alone. When there is constant suffering, there are certain places in which the pain is felt—at the points of emergence from the bony foramina of the different divisions, and where certain filaments become superficial. The frontal and supra-orbital, the infra-orbital, and the mental, are examples of the first class, and tenderness and pain are developed by pressure on the nerves at these foramina. These are nearly if not quite constant ; but those are less so, felt at the points where the nerves become superficial. The pains radiate from the painful points in both directions, but chiefly toward the periphery, and from the center, on other nerve-trunks—on the pneumogastric, on the occipital, etc. The sensibility of the part, innervated by the affected nerve, is altered; there may be merely perverted sensations, tingling, formication, etc., or anæsthesia when the case is old, hyperæsthesia when the attacks are recent. Photophobia, amblyopia, blepharospasm, and spasms of the facial muscles occur during the paroxysms. Various vaso-motor disturbances ensue, such as herpetic eruptions (zoster), eczema, falling out of the hair, a glossy state of the skin, ophthalmia, in old cases, and in the recent attacks, injected conjunctiva, lachrymation, swollen face, thickened skin, injected nasal mucous membrane, etc. When paroxysms are brought on by eating, and when sleep is prevented, the general health declines, but otherwise there may be no constitutional symptoms. Tic-douloureux may occur in one or all divisions of the fifth ; more frequently it is either confined or is most violent in one of these divisions. When the ophthalmic division is affected, pain extends into the forehead and temples, the eyelid, and the eye itself. The principal painful spot is at the supra-orbital foramen ; there is considerable hyperæmia of the conjunctiva, photophobia, and spasm of the orbiculus palpebrarum. When the second division is attacked, the pain is felt in the superior maxilla, in the teeth, and the upper lip. The principal tender point is at the infra-orbital foramen. When the third or inferior maxillary division is attacked, the pain is felt in the lower jaw, and in the teeth, and the most certain painful point is the mental foramen.

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* "Neuralgia and its Counterfeits," London, 1871, p. 31.

Course, Duration, and Termination.—Tic-douloureux may be several years in its development, attacks of pain becoming gradually more severe, better defined, and paroxysmal. It is therefore a chronic disease. That form dependent on malarial infection occurs more abruptly, has distinct periodicity, and terminates promptly, if appropriately treated, or assumes some other form. If caused by an aneurism, or tumor, or exostosis, the course is slow but usually uniform, and the pain and hyperæsthesia are excessive ; but after a time anæsthesia occurs and the pain declines. In the purely neuralgic form there is no regularity in the paroxysms, and a state of the peripheral nerves is ultimately reached when paroxysms are induced by the slightest movement. In the rheumatic subject, changes of temperature and barometric pressure may determine attacks which can be predicted. The simpler forms may terminate in recovery, but those cases due to exterior pressure on the trunk of the nerve within the cranium are incurable. Severe and protracted cases may terminate in epileptic attacks, or induce insanity, or lead to suicide.

Diagnosis.—To determine the cause of the neuralgia may be very difficult, and to separate the cases purely neuralgic from those due to some intra-cranial growth may be impossible at the outset. There is no difficulty in diagnosticating the seat and character of the neuralgia, apart from the lesion producing it. An intra-cranial growth affecting the nerve will be accompanied by other sensory and motor disturbances—by strabismus, double vision, vertigo, incoördination, paralysis, etc.

Treatment.—In cases produced by some form of infection, syphilitic, rheumatismal, plumbic, or malarial, treatment must necessarily be directed to the underlying cause. In every case in which no explanation is possible of the origin of the disease, it is good practice to prescribe a course of iodide of potassium. For the relief of recent cases, beginning suddenly and with violence, full doses of quinine and morphine (gr. xv—gr. xx of quinine and gr. ss. of morphine) are to be commended. Duquesnel's aconitine in solution, internally, in from $\frac{1}{100}$ grain to $\frac{1}{20}$ grain, even $\frac{1}{10}$ grain very cautiously, has been successful in some cases of pure neuralgia of the fifth. Fluid extract of gelsemium has had a curative effect in some cases, and a palliative effect in others. It should be carried to the point of inducing ptosis, dilated pupil, and muscular languor. To afford relief, there is no remedy comparable to the subcutaneous use of morphine, and this relief may be permanent, but is not frequently so, and the danger of inducing a morphine-habit is very great in a disease of this kind. The combination of morphine and atropine is preferable to morphine alone. Atropine hypodermatically has effected a cure in some cases. These remedies, if continued for a great while, lose their effect, and the pain which they at first relieved seems to be caused by them at last. Injections in the vicinage of the diseased nerve have been used with success. Water has been so used, and has afforded some relief. Of all the remedies thus far proposed none have been so successful as the deep injection of chloroform. This method is adapted to those cases of neuralgia in nerves superficially placed, as the supra- and infra-orbital nerves, because the chloroform must be deposited about the nerve or in its neighborhood. The author has published some cases showing the extraordinary relief, lasting months, and permanent cures which have thus resulted. The method consists in depositing in the neighborhood of the nerve from five to ten minims of pure chloroform by means of the hypodermatic syringe. The constant galvanic current, by the polar method, always affords great relief to the pain, and may in purely neuralgic cases bring about a cure. Daily applications of a few minutes should be kept up for a long time if improvement continues. Means to promote the nutrition of the body are important, for in neuralgia the vital forces are usually depressed. If anæmia exists, iron is necessary. Arsenic is one of the most powerful of the so-called nerve-tonics, and is particularly serviceable when indigestion exists. The phosphates and cod-liver oil are highly useful in the tic-douloureux which succeeds to lactation, or in all conditions of bodily depression. Nerve-stretching in this as in other forms of neuralgia is an expedient which should be tried when milder means fail.

CERVICO-OCCIPITAL, CERVICO-BRACHIAL, INTERCOSTAL, AND LUMBO-ABDOMINAL NEURALGIA.

Pathogeny and Symptoms.—The *cervico-occipital neuralgia* is situated in the region innervated by the four upper cervical nerves. The pain is felt in the occipital region to the vertex and ear, the neck downward to the clavicle, and upward and forward to the cheek, but chiefly in the distribution of the occipital nerve. The pain may occur on one side or both, but usually on one, is deep, heavy, and tensive, or sharp and lancinating, is paroxysmal, severe, and is increased by every movement, so that the head is held rigidly in one position. The course of the occipital nerve is tender. Hyperæsthesia of the skin and cramps in the cervical muscles occur, and attacks of herpes are common.—*Cervico-brachial neuralgia* arises under the same conditions as the other forms. The pain is very severe, of a boring, burning, heavy, and tensive character, and is usually very severe at night. The pain is accompanied by a sense of numbness, and weakness of the arm and hand, and is most severe in the shoulder and arm, but it extends down as far as the inferior angle of the scapula, and is often very strong in the mamma of the same side. The cervical plexus is very tender, and painful points are felt behind the acromion process, at the outer part of the insertion of the deltoid, over the median and ulnar, etc. The spinal apophyses, corresponding to the origin of the nerves implicated, are tender. Besides the pain developed by pressure, the skin of the arm at various points is hyperæsthetic, notwithstanding the numbness.

The arm feels heavy and useless, and power is actually impaired. At the outset, the arm is swollen somewhat, hot and rather red, but in an advanced case it shrinks from disuse, becomes pale, the skin glossy, dry, and harsh.—*Intercostal neuralgia* is produced by causes besides those of the other forms of neuralgia. Aneurisms and tumors of the chest cause very violent attacks of pain. Diseases of the vertebra and ribs have the same effect. The pain is of two kinds—a feeling of soreness with fatigue, and an acute lancinating pain. As in the other forms of neuralgia, the pain is paroxysmal, remits and even intermits. Pain in the left side, usually referred to the sixth or seventh intercostal space, is very common in women, and is apparently due to ovarian and uterine irritation. Intercostal neuralgia not unfrequently takes the form of *herpes zoster* or shingles. The author has seen eight cases in which the herpes seemed to be due to arsenic, and others have made the same observation, so that the assumption, that, when zoster accompanies intercostal neuralgia, neuritis is the cause of both phenomena, seems hardly justified. In young persons there is not much neuralgia with zoster, and, in the old, the neuralgia precedes and succeeds the eruption. In most cases there is a burning pain which comes on just as the eruption is about to appear, and also acute lightning-pains shooting through the chest.—*Lumbo-abdominal neuralgia* includes the ileo-hypogastric nerve, the ileo-inguinal, and the external spermatic nerve supplying the hypogastrium, integument of the hip, the inner face of the thigh, and the scrotum or labium, but neuralgia of these nerves is rather uncommon.

SCIATICA.

Definition.—The sciatic plexus is made up of the fourth and fifth lumbar and the first two pairs of sacral nerves. The term *sciatica* is applied to a neuralgic affection of the sciatic nerve. Sciatica is, next to tic-douloureux, the most important of the neuralgic affections.

Pathogeny and Symptoms.—Constitutional predisposition and heredity have less to do with sciatica than with any of the other forms of neuralgia. The disease occurs much more frequently in men than in women. Direct injury to the nerve in certain positions—sitting, especially if the form of the seat is such as to direct the weight of the body on the nerve; by prolonged walking; by constipation, the bowel being distended with hardened fæces—is the most influential cause. To these must be added exposure to cold and dampness, as, for example, prolonged sitting on a damp stone, fatiguing work in the standing posture in water, etc. These causes are the more influential if the system is predisposed by rheumatism and other cachexiæ and by the neuropathic constitution. It may be stated, in general terms, that sciatica is produced by the same causes, constitutional, immediate, and remote, that other forms of neuralgia are, but that it is much more likely to be developed by local and mechanical than by systemic and constitutional causes. The only pathological alterations proper to sciatica are those of neuritis. As a result chiefly of disuse, the affected limb wastes more or less in severe cases. The disease develops slowly. In most of the cases observed by the author, an attack of lumbago preceded the sciatica, and the pain gradually became fixed in the sciatic. In several cases (four) the pain began in the heel. In other cases the first symptom noted was a feeling of pain and soreness in the hip. A feeling of stiffness, numbness, formication, heaviness of the limb, and other abnormal sensations have been noted. In what way soever the disease begins, soon severe pains occur in distinct paroxysms. The pains are lancinating, tearing, grinding, and they shoot with lightning-rapidity along the direction of the principal nerves. Now they are felt with greatest intensity in the hip behind the joint, again in the calf of the leg, now in the ankle, again in the heel, or the pain flies from one to another of these parts, or shoots through them all at the same time. The paroxysms last a variable period from an hour or two to twenty-four or more hours, sometimes for several days, there being brief remissions only. The pain is almost always worse at night. In the interval between the paroxysms the limb is heavy, movements excite pain, and there is a tensive, throbbing sensation which threatens severer suffering. Exercise usually increases the pain, and unguarded movements may bring on a paroxysm. The trunk of the nerve behind the trochanter is sensitive to pressure, also in the popliteal space; there are tender points at the head of the fibula, behind the inner malleolus and also behind the outer malleolus, and there is tenderness of the lumbar apophyses. The pain often radiates into the lumbar nerves, into the sciatic of the opposite side, and into the scrotum and testes. Hyperæsthesia and cramps occur at first, and in old cases diminished sensibility, lowered temperature, and wasting are observed. The appetite is impaired, there is little sleep in bad cases, and hence the bodily forces decline. At first the limb is used awkwardly, the patient limps, then crutches are resorted to, and finally the bed is the only resource. The pitiable state to which a man can be reduced by a severe sciatica is told by a sufferer, himself a physician, Dr. Lawson:* "The pain persisted for more than six months; it first reduced me to the employment of crutches, and then absolutely prevented locomotion; the limb became permanently flexed and terribly wasted; nearly every remedy in the Pharmacopœia, and many out of it, were tried in vain; . . . for six months I had hardly known what sleep was, notwithstanding the administration of opiates three or four times a day. Appetite was utterly lost; physical power was prostrate ; mind, through long suffering, was enfeebled to that degree that I look back upon that period of my existence with astonishment and horror." Of course, not all cases are so severe as this of Dr. Lawson, but in every mild case suffering is experienced, the sleep is broken more or less, but the general health does not suffer any considerable deterioration.

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* "Sciatica, Lumbago, and Brachialgia, etc.," by Henry Lawson, M. D., London, 1872, p. 7.

Course, Duration, and Termination.—After the first acute symptoms, when the case begins with lumbago and a feverish state, the course is chronic and like the usual pattern. When the symptoms develop slowly, the disease reaches its maximum in a few days, or a week or two. If the treatment be appropriate, a termination in health may take place in two or three weeks. The cases often continue months and years, in varying condition, now improving, then getting worse. In the author's experience, there are two climatic states which exercise an unfavorable influence—variable cold and damp weather and continued high temperature ; while uniform dry cold has a favorable effect. Quite irrespective of climatic changes, sciatica has a strong tendency to relapses. Some cases gradually subside without any properly directed treatment, and get well in a year or two. Many do not recover entirely, although there may not occur any acute paroxysms ; the limb continues weak and a halting gait persists, because of imperfect combination of the muscles. Cases occurring in old subjects, whose symptoms present the evidences of senile degeneration, may continue during life.

Diagnosis.—Ordinarily a case of sciatica does not offer any difficulties for careful consideration. It may be confounded with muscular rheumatism, with the first stage of hip-joint disease, and with hysterical joint. Muscular rheumatism differs from sciatica in the lesser severity of the pain, in the absence of distinct paroxysms, and in the diffusion of the symptoms, the distress in the one being distributed over the principal muscles, in the other confined to the nerve-trunks and to certain painful points. In incipient joint-disease there may be much sciatica, so that the distinction must rest on the changes in the shape of the hip, in the gluteal fold, and in the position of the foot, which, with the history, ought to indicate the existence of hip-joint disease. The hysterical joint is differentiated by the absence of any evidence of suffering, by great tenderness in the skin, and yet, when the attention is withdrawn, by entire lack of tenderness in the nerve-trunk or in tender points, and by the evidences of hysteria present.

Treatment.—Existing causes should be removed. If the attack depends on impaction at the flexure or cæcum, active purgatives should be prescribed. A particular chair or habit of sitting may be responsible, and should be changed. If the attack begin by lumbago, warm baths, Russian or Turkish, may soon effect a cure. Dr. Lawson, whose shocking experience has been referred to, after six months of unavailing treatment, was at once relieved and speedily cured by the hypodermatic injection of morphine. His little work, written to advocate this treatment, contains numerous cases illustrating its utility. Morphine (gr. $\frac{1}{6}$ to gr. $\frac{1}{4}$) and atropine (gr. $\frac{1}{120}$ to gr. $\frac{1}{100}$ to gr. $\frac{1}{60}$) are more effective in combination than morphine alone. The injection is somewhat more effective when inserted in the neighborhood of the affected nerve. There can be no doubt that this treatment is sufficient in itself in many cases, but it can be aided by other measures, local and systemic. The author has witnessed remarkable cures of chronic cases by the deep injection of chloroform. This practice consists in the injection of five to ten minims of chloroform, thrown deeply in the neighborhood of the nerve near to the point of its emergence from the pelvis. The injection should also be practiced at those points where the pain has been severe. But few injections are necessary. Ether may be used also, but it is more irritating and less effective. The author has cured many cases by stabile applications of galvanism alone. A large sponge electrode should be applied over the nerve near the point of exit from the pelvis, and the other electrode below. Strong currents are more effective and, indeed, indispensable for curative results. Successive portions of the nerve should be included in the circuit, by applying the anode over the painful points and the cathode below, according to the method of Remak.* Eulenberg,† Erb,‡ and Althaus, are fully agreed as to the success of the galvanic current in sciatica. Hammond has revived the method of Magendie, and now cures sciatica by inserting an acupuncture needle, insulated to near its end, and passing through it a current from a few cells. Firing is often very successful. The hammer, dipped in boiling water, is applied to produce redness and slight vesication, or considerable burning, according to the duration of the case. Great relief and even curative effects have followed the application of blisters, the raw surface dressed with powdered morphine. Flying-blisters are beneficial. The warm pack and the rubbing pack are of great service in obstinate cases. The pack may be worn all night. In the chronic cases of supposed rheumatic origin, iodide of potassium, guaiacum and turpentine are said to be useful, but the author has not seen any good results from them. The other forms of neuralgia referred to above require the same treatment. Any local injury, constitutional condition, or cachexiæ, must be removed. The most successful remedies are the hypodermatic injection of morphine and the constant current, the curative influence of which few cases resist.

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* "Galvanothérapie, traduit de l'Allemand par le Dr. Morpain," Paris, 1860, p. 374.

† "Lehrbuch der functionellen Nervenkrankheiten," *op. cit.*, p. 168.

‡ Ziemssen's "Cyclopædia," vol. xi.

SPASM OF THE FACIAL MUSCLES SUPPLIED BY THE SEVENTH NERVE—CONVULSIVE TIC—HISTRIONIC SPASM.

Definition.—The seventh nerve is distributed to the muscles of expression. The attacks of spasm may occur in all or a part of these muscles. *Convulsive tic* or *mimetic spasm* is the term applied to the former ; *blepharospasm* is the name given to spasm of the eyelids.

Pathogeny and Symptoms.—Various causes are assigned for the production of mimetic or histrionic spasm. The constant activity and variety of movement in expressing the various emotions render these muscles rather apt to take on abnormal movements. This is seen in tricks of expression imitated from others, and also inherited, but the direct transmission of histrionic spasm is not common. Men are more apt to suffer from this malady than women. It may occur as a secondary symptom in such convulsive disorders as chorea, epilepsy, etc. It may be developed from purely psychical states, as anger or fear, but then a predisposition must exist. It is more apt to arise from direct or reflex irritation of the facial nerve. Tumors, caries of the bones, diseased teeth, periostitis, and remote irritation, as intestinal worms, have set up the spasms. The disease begins in a small group of muscles, and then extends to all the muscles, on one side usually, although both sides may be affected. It consists in a succession of clonic spasms, producing extraordinary grimaces and contortions. If one side, it is all the more striking by comparison with the unmoved state of the unaffected side. The spasms occur in paroxysms, lasting a few seconds or a few minutes. They begin in one group of muscles by a few twitches, and then clonic spasms follow in all the others. It is a rule, however, for the attack to be more decided in some one muscular group, as in the orbicularis palpebrarum and corrugator supercilii, and levator labii superioris et alæque nasi and levator anguli oris. The number of the attacks varies greatly, usually several occurring every hour, and they may persist during the night, but this is not usual. They are excited by attention to them, by talking, by emotion, and by increased irritation of the nerve-trunk. They do not interfere with the normal use of the muscles at other times. Extension of the spasm may take place to the muscles of the tongue and to those of mastication, and in severe paroxysms the muscles of the neck and shoulders may participate. The electro-contractility of the muscles remains unaffected. *Blepharospasm* is the form of the disease attacking the eyelid. This consists of paroxysmal attacks of sudden closure of the lids, with spasms of the annexed muscles, producing extraordinary grimaces of the affected eye. The attacks may occur suddenly without any apparent cause, or be induced by straining or irritation of the eyes, by opening or closing the lids. The conjunctiva is injected, there is a profuse secretion of tears, and an extreme degree of photophobia may exist. These changes may be the result of blepharospasm, but, in a great majority of cases, diseases of the eye, as scrofulous conjunctivitis, corneitis, wounds, by irritating the sensory fibers of the fifth, excite the spasms by a reflex mechanism. In this disease certain so-called *pressure-points* exist, pressure on which will suddenly arrest the paroxysms. These have no fixed position, as the painful points in neuralgia, and can not be indicated beforehand in any case, but must be searched for. They are sometimes found at the supra-orbital foramen, and on various branches of the fifth nerve in the face, the gums, the malar bone, and the mastoid process, and if not detected in these situations may be discovered in the brachial plexus, the spinous processes, or the sympathetic. Pressure on these points exerts an inhibitory influence on the spasms, which may be suspended for some time. On the other hand, the influence of the pressure-points may continue only during the pressure (Erb).

Treatment.—The removal of any cause of irritation, intrinsic or extrinsic, is necessary. As blepharospasm is so often due to strumous diseases of the eye, these must be removed before any influence can be exerted on the spasm. Remarkable results have been obtained from the free use of *succus conii* in this malady; in recent cases, the subcutaneous use of morphine, and morphine and atropine. The hypodermatic injection of Fowler's solution has succeeded remarkably in some cases of tic. From two to five drops can be injected daily about the pes anserinus. The constant current (stabile) applied to the pressure-points, the positive pole on the point, the negative held on some part of the periphery, has been successful in some cases. The sympathetic, the mastoid process, the vertebræ, etc., are also possible pressure-points to which the current should be applied. Remarkable results have followed the section of the supra-orbital nerve in a few cases.

SPASM OF THE MUSCLES SUPPLIED BY THE SPINAL ACCESSORY—TORTICOLLIS.

Pathogeny and Symptoms.—The trapezius and the sterno-cleido-mastoid are the muscles affected either separately or together, and the attack may be unilateral or bilateral. In unilateral spasm of the sterno-cleido-mastoid, the head is rotated a little, the chin elevated and turned to the other side, and the occiput is brought forward and downward in the direction of the clavicle. If the trapezius is alone affected, the head is drawn down and backward, and the shoulder upward and inward toward the spine. When both muscles are affected, there is a combination of the movements, and they may alternate. In bilateral spasms of the spinal accessory, the head is drawn from one side to the other, and the chin correspondingly turned in the opposite direction. If the sterno-mastoids are alone affected, there occur symmetrical nodding movements. The attacks of spasm are paroxysmal, and are of variable duration, lasting from a few minutes to a number of hours. They may be very severe, tossing the head from side to side in a terrible manner, and may be almost continuous, involving also the muscles of the face, of mastication, and of the shoulder. Sleep usually arrests the movements, and is quiet and undisturbed, although it may be delayed, and sometimes entirely prevented. The paroxysms are excited by any kind of irritation, as of talking, mental excitement, anger, and are increased by the attention given to the spasms by others. As a necessary result, the wild, disorderly, and very strong movements exhaust the muscles. In the course of the paroxysms, speech and mastication are prevented. The unpleasant condition of these patients and the nervous disorder probably associated with it slowly bring about a mental change. These patients are depressed and gloomy, sometimes suicidal, and, in the further progress of the case, epilepsy, paralysis, or insanity may be a result.

Treatment.—There is little to encourage therapeutic effort, and partly because the origin remains obscure. Those cases brought on by exposure of the neck to draughts of cold and damp air are the most remediable. If there be a source of reflex irritation which can be removed, as worms, indigestion, or uterine disease, the muscular disorder may be readily cured if treated in time. When there are intra-cranial lesions, or if the case be chronic, and occurring in the neuropathic constitution, the treatment is in vain. The best results are obtained from the constant galvanic currents, stabile applications, and by applications to the sympathetic and to the spine. Next in efficiency is the hypodermatic injection of morphine, if possible, into the muscles affected. The injections of arsenic should be tried in doubtful cases. The warm pack should be steadily worn at night, and douches to the cervical spine applied warm or cold, according to the results. The actual cautery has been used with success in a few cases. In that form of *torticollis* in which the muscles assume a condition of tonic spasm, they are fixed in a permanent position by contraction. If the sterno-cleido-mastoid is affected, it stands out prominently and is enlarged and rigid, and the head assumes a characteristic attitude, the chin turned away, and the occiput brought down and forward toward the clavicle. When the trapezius is alone affected, the head and shoulder are approximated, and the anterior border of the muscle forms a prominent, rigid swelling. The affected muscles have a sore, tired feeling, and are tender to the touch when the affection is recent. The antagonistic muscles after a time undergo atrophy, and hence the overacting muscles are aided in maintaining the fixed position of the head. In young spines a permanent curvature of the cervical part takes place, and the features accommodate themselves to the changed position of the head in a most remarkable way. The bones of the face undergo a slow transformation to permit the features to assume the new relations. In this disease it is highly important to undertake the treatment before the deformity becomes permanent. Electricity is entitled to the first place as a remedy. There are two methods of application to be employed. Stabile applications are to be made to the muscles in a state of spasm, and faradic currents to the antagonistic muscles. Warm packs, massage, and gymnastic training are useful. Surgical treatment is necessary in chronic cases.

SPASM OF THE DIAPHRAGM—SINGULTUS—HICCOUGH.

Pathogeny and Symptoms.—This malady consists in a recurring spasm of the diaphragm; there is first a full expiration, then a sudden inspiration, accompanied by a high tension-sound, caused by a spasmodic closure of the glottis. It is often present without having any significance. It is a symptom of certain kinds of indigestion, and is present only during the stage of digestion. Distention of the stomach may cause it. Hepatic diseases—peritonitis, chronic ileocolitis—are maladies during the course of which hiccough may come on, especially in the collapse which ushers in death. It is a symptom of irritation of the respiratory center, and of various diseases of the central nervous system, and is one of the manifold forms in which hysteria manifests itself. The worst case ever seen by the author occurred after a severe attack of hepatic colic. When the paroxysms are protracted and the hiccough is frequent, very considerable suffering is the result. The hiccough may occur as often as one hundred to the minute, and the paroxysms may continue for some hours or days, returning from time to time during several years. The attacks may have a certain rhythm, three, six, or other numbers occurring in succession, then an intermission. When a severe paroxysm comes on, severe pain is felt in the epigastrium, the respiration is disturbed, eating is difficult, and sleep may be prevented.

Treatment.—A strong mental impression or a draught of very cold or very hot liquid will sometimes succeed in arresting hiccough. Electricity is usually very successful. In the severe case just mentioned the author arrested the spasm instantly, after all kinds of remedies, including galvanization of the phrenic, had been tried in vain, by sending a strong faradic current through the diaphragm just as the spasm was about to occur. The inhalation of ether, of nitrite of amyl, and the injection of pilocarpine, have all promptly succeeded.

PARALYSES OF THE OCULAR MUSCLES.

Pathogeny and Symptoms.—Paralysis of the muscles of the eye is a symptom rather than a disease. Rarely does a case happen in which the paralysis is due to rheumatic inflammation. More frequently penetrating wounds, contusions, and fractures, are causes. The secondary paralyses are more numerous than the primary. Diseases of the brain, such as cerebral hæmorrhage, tumors so situated as to compress the nerve-trunks, affections of the spinal cord, as posterior spinal sclerosis, and the paralysis following diphtheria, are the most influential causes. When the muscles are weak, the movements of the ocular globe are affected, a fact which may be made apparent by comparing the sound with the impaired eye ; the limit of rotation will be seen to be less, and the obvious result is strabismus. Before this is apparent by ordinary inspection, the patient complains of diplopia (double vision). Or there is confused double vision, the patient being affected only in certain parts of the visual field. The secondary deviation of the sound eye is a very characteristic sign. "The field of vision is displaced in the direction of the action of the paralyzed muscle," which leads to erroneous perception of the position of objects. The disturbances of vision caused in this way induce giddiness and more or less pain. Covering the eye prevents, of course, the formation of a double image, and thus affords some relief. When the *motor oculi* is paralyzed, there is *ptosis* (dropping of the eyelid), and the movements of the eye downward, inward, and upward, are lost. The pupil is dilated and motionless because of the unopposed action of the sympathetic, and the power of accommodation to near and distant objects is very much lessened. As the external rectus and superior oblique continue in action, the eye becomes fixed in the direction downward and outward. The eye is usually prominent because of the paralysis of the straight muscles, allowing the globe to glide forward. There is double vision, and, as the field of vision is falsely projected in every direction, there is great disturbance of visual perceptions, and consequently giddiness, so that the eye is ordinarily kept closed. In paralysis of the abducens, the external rectus muscle is unable to move the eye outward, and there is consequently convergent strabismus.

Course, Duration, and Termination.—There are very great variations in the course of these affections, as they are dependent on various causes. The rheumatic affections may be regarded as curable with comparative facility, but those examples due to intra-cranial lesions, unless syphilitic, pursue the course of the original disease, and are incurable. The accompanying symptoms are of great importance in coming to a conclusion as to the seat and character of the local disease.

Treatment.—If syphilitic, rheumatismal, or plumbic lesions be the cause, the treatment appropriate to these diatheses should be carried out. In the absence of any specific cause, a course of the iodide of potassium should always be undertaken. The most important remedy, and one from which most striking results are obtained, is electricity.

Labile applications of galvanism are the most effective—the anode placed on the mastoid, and the cathode passed over the eyelids. The current must be strong enough merely to cause movements of the facial muscles, and the length of the sitting should be about three minutes. The sympathetic may also be galvanized in the usual way. The faradic current, which is greatly more painful, may be used instead in some cases—one pole on the temple, and the other, covered with soft leather, to the conjunctiva at the situation of the paralyzed muscle, if possible.

PARALYSIS OF THE FACIAL NERVE—FACIAL PARALYSIS.

Causes.—Exposure to a current of cold air, directed against the main divisions of the nerve in front of the ear (pes anserinus), is the most usual cause, and of the simplest variety of the disease. Such exposure acts by exciting some inflammation of the neurilemma; in the Fallopian canal serous and occasionally plastic exudation occurs and compresses the nerve. Injuries to the nerve in front of the ear are very common, but the most usual cause, next to cold—the so-called rheumatic inflammation—is disease of the middle ear. Syphilitic deposits, gummata, etc., may invade the nerve before its entrance into the canal, and also various diseases of the basal ganglia, tumors, exostoses, etc. Again, facial paralysis occurs with hemiplegia, or it may be a crossed paralysis in disease of the *pons Varolii.*

Symptoms.—No disease is more distinctive than facial paralysis. The affected side is perfectly blank, motionless, without wrinkles, the corner of the mouth depressed, the eye wide open, and the tip of the nose and the whole side drawn over to the healthy side, which is more strongly marked by furrows and wrinkles than before. This condition of the muscles may occur suddenly: the patient, on looking in the mirror in the morning, is astonished and alarmed at the change; or, feeling an odd sensation in the lips and tongue, he attempts to expectorate or whistle and finds he can not use his lips properly. There may be premonitory symptoms for some hours, even a day or two before the attack, consisting of numbness and tingling of the lips, a strange taste, acid or metallic, pains in the face or ear-ache, noises in the ear, or there may be present an otorrhœa. Again—and this is especially true of disease of the middle ear—the paralysis may develop slowly, one group of muscles, then others, becoming paralyzed, and, when complete, all of the muscles innervated by the seventh nerve are affected. When this occurs, no movements can be effected by these muscles. The eye remains open; the conjunctiva inflames in consequence of the particles of dirt which alight and adhere; there is a profuse flow of tears; in attempts to close the eyes, the upper lid falls and the globe rotates upward and inward, but the lids do not approximate, and hence the eye remains open, and in time the lower lid becomes somewhat everted;

the forehead can not be corrugated. The corner of the mouth can not be elevated, the lips can not be pursed up in the attempts to whistle, and in smiling the affected side remains motionless, while the sound is acting strongly. The saliva escapes from the mouth, and the labials can not be pronounced, whence the speech is rather mumbling and indistinct. Mastication is difficult and the alimentary bolus accumulates in the cheek of the paralyzed side. Not unfrequently the sense of taste on one side of the tongue is abolished, and the secretion of saliva lessens. When this is the case, the chorda tympani, which Schiff has shown is the nerve of taste to the anterior half of the tongue, is affected, and it therefore follows that the seventh is damaged at the point of origin of this nerve. The uvula is often affected also, and hangs paralyzed, deviating toward either side. When this organ is affected, the speech is nasal, swallowing is difficult, and liquids come through the nose. This paralysis of the uvula is necessarily due to implication of the superficial petrosal nerve. The ear is usually unaffected, although noises are heard. The sensibility of the paralyzed side is normal. The reflex movements are entirely abolished when the disease occupies any part of the trunk of the seventh from its origin outward. In case of hemiplegia the reflex excitability is preserved. In the mildest cases the electro-sensibility and contractility are perfectly normal. In the more severe cases the muscles may not respond to a faradic current, yet do respond to a slowly interrupted galvanic current; but the nerves themselves lose their excitability to both currents during the period of regeneration. The muscles may ultimately lose their galvanic excitability when they have undergone advanced changes. When this is the case, the prognosis is unfavorable.

Course, Duration, and Termination.—When the external branches of the seventh only are affected, and by such a simple cause as exposure to a current of cold air, the duration will be short, and recovery effected in two or three weeks. The more severe cases may require twice the time of the former. In those cases characterized by loss of faradic and retention of galvanic excitability of the muscles, the duration will be several months, even a year may elapse before restoration. In these cases, after a time, the muscles become rigid and retract somewhat, and they may be affected by spasmodic contractions resembling tic. In traumatic paralysis, the amount of recovery depends on the extent of injury to the nerve. Usually restoration in the most favorable cases is incomplete. The same observations may be made of paralysis from pressure of the nerve, the degree and curability of injury determining the result.

Diagnosis.—The diagnosis is reached by mere inspection, but to ascertain the seat of the injury to the nerve is more difficult. Whether peripheral or central is arrived at by attention to the following points: in peripheral paralysis, the eye is wide open even in sleep, and reflex movements of the lids are abolished, which is not the case in cerebral paralysis; the abolition of faradic and the retention of galvanic excitability and the degeneration of the muscles—symptoms not present in the cerebral form; in the latter are observed various cerebral symptoms. The position of the disease in the trunk of the nerve may be determined as follows: paralysis of the muscles of the face, without involving taste, indicates with other symptoms disease of the nerve anterior to the origin of the *chorda tympani;* paralysis of the muscles, no reaction to faradic but response to galvanic current, paralysis of uvula, indicate lesion of the nerve at the origin of the large superficial petrosal nerve which goes to the spheno-palatine ganglion. When there is alternating paralysis, the lesion is most probably in the pons. If partial paralysis exist, the velum palati being affected at the same time, and if the reflex and electrical excitability are preserved, the lesion is in the opposite hemisphere of the brain or its crus.

Treatment.—The cause of pressure on the nerve within the cavity of the cranium, or disease of the ear, should be removed if practicable. In all doubtful cases a course of iodide of potassium should be prescribed. If the attack is of the rheumatic variety—so called—blisters to the mastoid and the internal use of pilocarpine are the most effective measures. The application of electricity, the galvanic current preferably, should be begun at once, and continued faithfully until a cure is effected or discovered to be unattainable. The application should be made by one pole—the anode—on the mastoid, and the cathode passed over the terminal filaments of the nerve as distributed to the muscles.

VASO-MOTOR AND TROPHIC NEUROSES.

HEMICRANIA—MIGRAINE.

Definition.—By the term *hemicrania* is meant a unilateral pain in the head, irregularly periodical, and accompanied by nausea and sometimes vomiting, and excited by certain reflex disturbances. By the French writers it is termed *migraine,* which has been naturalized to a large extent in our country, and it is known in common language as *sick-headache.*

Causes.—Regarded by Romberg as an hyperæsthesia of the brain, the localization of the disturbance in the vaso-motor system was first distinctly affirmed by Du Bois-Reymond, who maintained that the cause of the affection is a contraction of the arterioles on the affected side of the head—a fact determined by observation on himself. An opposite view of the state of the sympathetic was taken subsequently by Möllendorff, who maintained that the vessels are relaxed. As is often the case, the truth probably lies between these extremes, as Eulenberg maintains. Females are more liable than males, and in early life the disease first manifests itself. It is distinctly inheritable, or at least the neuropathic constitution.

Symptoms.—The disease is irregularly paroxysmal, and in the interval between the attacks there is no pain or other disturbance. The paroxysms may or may not be preceded by prodromal symptoms, such as weariness, hebetude of mind, etc., but the onset of the attack is usually announced by chilliness, nausea, yawning, and general muscular soreness. The pain comes on most frequently on the left side, and is felt in greatest intensity in the supra-orbital ridge and in the eye, but it may be felt nearly equally over the whole side, and even extend over beyond the median line; usually there is a region of greatest severity of pain. Tenderness is felt when the cervical ganglia—upper and middle—are pressed on, and tenderness is also experienced when the spinous processes of the last cervical and first dorsal vertebræ are subject to pressure. The sense of touch is more acute than normal over the whole area of the hemicrania. In many subjects nausea and vomiting precede the attack of hemicrania; in others the pain continues for some time before nausea is experienced, and vomiting often ends the attack. Light is hurtful to the eyes, and noises to the ears. Rings of light and muscæ volitantes float before the eyes, and there are noises in the ears. The circulation, temperature, and secretions of the affected part are altered. There are, as Eulenberg insists, and as the author has repeatedly observed, two kinds of disturbance in the circulation: contraction of the vessels, and anæmia of the affected part, as shown in pallor of the face, shrunken eye and dilated pupil; dilatation of the vessels, flushed and red face, the conjunctivæ injected, and the pupil contracted. The two forms may coincide, but this is rare, and there may be cases in which no disturbance exists in the sympathetic ganglia.

Course, Duration, and Prognosis.—The paroxysms may last for a few hours or a day or two. They may occur every few days, every week, or every month, or at longer intervals. Women are especially liable to attacks about the menstrual period. In many they are induced by errors of diet. As the pneumogastric nucleus lies alongside of the nucleus of the fifth, it is easy to understand the transference of sensations. Usually the susceptibility to attacks declines with the advance in life and disappears after fifty. The author has frequently observed that the disappearance of hemicrania has been coincident with the occurrence of cerebral hæmorrhage. Otherwise, the disease must be regarded as entirely free from danger to life, while its chief importance lies in the fact that few cases are permanently cured.

Treatment.—The most important point is a careful regulation of the diet in that large proportion of cases originating in stomachal disorder. An easily digested aliment of the nitrogenous kind, with decided diminution of the farinaceous and saccharine elements, is the kind of diet required. In these cases the best results are obtained from the use of arsenic—two drops of Fowler's solution before each meal, kept up for months. In the other group of cases, nervous in origin, the best remedies are cuca, guarana, caffein, and bromide of potassium. The last mentioned is adapted to those cases dependent on contraction of the arterioles, and is very effective if administered just before the onset of the paroxysm, in a sufficient dose (ʒ ss. — ʒ j), and repeated several times. The other remedies mentioned are better fitted to give tone to the sympathetic ganglia in the interval between the paroxysms. When there is anæmia, a chalybeate course is highly serviceable. When the moral surroundings are such as to cause attacks, change of scene is highly necessary. If the disposition to the malady is inherited, the prophylaxis is very important and should include diet, exercise, clothing, and the avoidance of all those conditions which tend to develop an abnormal excitability of the nervous system. The best results have been obtained from galvanization of the superior ganglia of the sympathetic; the positive pole over the ganglion and the negative on the epigastrium in the tetanic form; and the poles reversed in the paralytic form. Frommhold * has obtained the best results from the faradic current.

ANGINA PECTORIS.

Definition.—A neurosis of the heart, in which there occur paroxysms, characterized by pain in the præcordial region, extending usually into the left shoulder and down the left arm, and accompanied by a feeling of constriction of the thorax, and a strong sense of impending dissolution. It is sometimes called neuralgia of the heart.

Causes.—A predisposition to this affection seems to be inherited. It is often associated with chronic cardiac changes, as arteritis of the coronary artery, calcification of valves, etc. It is, as Trousseau first pointed out, sometimes a masked epilepsy, and again angina pectoris may alternate with epileptic attacks. It may occur in hysteria, and may precede an outbreak of mania. Males are greatly more liable to it than females, and, although it is more frequent in advanced life, it may occur at any age. Excessive smoking by young and nervous subjects may cause it at a comparatively early age.

Pathological Anatomy.—Various changes in the heart are found, but these are accidental. The pathological changes which stand in a causative relation to the attacks are those of the cardiac plexus of the phrenic and of the pneumogastric nerves. Pressure of enlarged lymphatics, inflammation of parts of the cardiac plexus, with changes in the coronary artery, seem to be the most constant (Eulenberg).

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* "Die Migraine und ihre Heilung durch Electricität," Pesth, 1868, p. 115.

Symptoms.—Angina pectoris is a paroxysmal affection, the attacks occurring irregularly, and in the interval there are no symptoms. The attacks are eminently characteristic. The patient is suddenly seized, it may be in the night, during exercise or while resting, with an intense pain in the præcordial region, accompanied by a sense of constriction and suffocation. He at once assumes a fixed position as if the least movement would cost him his life; his face becomes deadly pale, and a cold sweat bedews the skin. The pain shoots across the chest, upward under the sternum and toward the left shoulder, and down the left arm. The sudden pain and terror may cause syncope, but usually the pain ceases in a few seconds or minutes, and the patient takes a deep breath with a sigh of relief. The respiration may continue undisturbed, may be very much oppressed, or it may be arrested, simply from a fear that the least movement may end life. The pulse is small, the action of the heart weak or arrested, and the arterial tension very high. A decided contraction of the superficial arterioles causes the skin to assume a pallid appearance, and a sudden chilliness with chattering of the teeth occurs. When the attack is over, the circulation becomes active, the skin warm, eructation of gas, sometimes vomiting, occurs, and a quantity of pale, watery urine is passed.

Course, Duration, and Termination.—The course of the disease is chronic. The paroxysms have a variable duration—usually lasting a few seconds only, but they may continue, with remissions in the severity of the symptoms, for hours, even days. The return of the attacks is irregular and uncertain; they may appear after an intermission of days, or weeks, or months. It is usually several months after the occurrence of the first seizure until the next appears. The nocturnal attacks are spontaneous in origin, but those occurring during the day are caused by some strong emotion—a fit of anger, chagrin or disappointment—by some active exercise, or by indigestion. The disease may occur irregularly during five to seven years. The importance of angina is largely affected by the cardiac lesions which usually accompany it, and the fatal termination so often observed after two or three paroxysms, rarely in the first, is due to these associated cardiac lesions. Whether symptomatic or essential, angina pectoris is a fatal malady, but the latter form is more amenable to treatment, and offers a longer duration than the former.

Treatment.—All causes of disturbance of the cardiac action, as tobacco-smoking, etc., must be removed. Those attacks accompanied by vascular spasm—and this seems to be the case during the paroxysm in all cases—are most promptly relieved by the nitrite of amyl, originally proposed by Brunton. Patients should be provided with the perls containing three minims, to be broken in the handkerchief, and the vapor inhaled on the instant. This expedient has given relief in a large number of cases. The solution of nitro-glycerine has been used most successfully in the interval of the seizures to prevent them. Full doses of arsenic (ten minims of Fowler's solution) three times a day, after meals, have a good effect. The hypophosphites and cod-liver oil, continued steadily for months, have done good in debilitated subjects. Where a malarial influence may be presumed to exist, quinine is the proper remedy. When epilepsy is masked under attacks of angina, bromide of potassium affords great relief. Remarkably good results have been obtained from galvanism, stabile currents being used—the positive pole at the præcordia, and the negative over the seventh cervical vertebra.

EXOPHTHALMIC GOITRE (GRAVES'S DISEASE).

Definition.—*Exophthalmic goitre* is a disease characterized by a quaternary of symptoms—exophthalmus, enlarged thyroid, dilatation of the arteries, and palpitation of the heart. It has received a variety of designations. In Germany it is known as *Basedow's disease;* in England, *Graves's disease*, from the names of supposed discoverers.

Causes.—Although a variety of causes have been alleged, few are worthy of serious consideration. Heredity, anæmia, and chlorosis, moral emotions, have been considered causative, but of these only the last appears to have exerted any real influence. In the cases seen by the author, fright, chagrin, reverses of fortune, etc., were the causes, but it is probable that the effect produced was really due to some peculiar condition of the nervous system. This disease is more common in women than in men—in the former before, in the latter after thirty, whence it may be concluded that a mobile nervous system is necessary to its origin.

Pathological Anatomy.—The changes characteristic of exophthalmic goitre are by no means striking. The veins and arteries of the thyroid show great increase of size and thickness, and the gland itself is unaltered, or in the condition of simple hyperplasia, or cystic; but the last-mentioned state has no relation to this disease. A considerable increase in the fat behind the eye has been observed; the muscles are affected with fatty degeneration (one case); the ophthalmic artery is atheromatous (one case)—but these are probably only accidental changes. Some structural alterations have been found, in a majority of cases, in the sympathetic ganglia, and especially in the inferior ganglion. Both sides may be affected, or one only, and the amount of disease varies greatly. The heart in most, if not all, cases is damaged variously, but these changes are not a part of this disease, and are entirely accidental.

Symptoms.—In one of the author's cases the first symptom (protrusion of the eyes) was perceived by the patient on going to the mirror in the morning. She had been subjected to a great shock the previous evening. Usually the onset of the disease is gradual, so that there are really two types, the acute and chronic. The acute cases may run their whole course in a few months. The initial symptom may be any one of the four great characteristics, but palpitation is most often the first departure from health. The increased action of the heart is at first paroxysmal, with intermissions during which the rate is normal ; but the intervals shorten until the heart-beat is always above normal, with paroxysms during which marked acceleration takes place. When the acceleration attains its maximum, the ordinary rate is from 90 to 120, but during the exacerbations 160, even 200, may be reached. A soft-blowing murmur is usually audible at the base, and propagated along the great vessels, and a stronger, whirring, blowing murmur is to be heard over the carotids and the thyroid ; an epigastric and sometimes hepatic pulsation may be detected. The vessels of the neck and of the thyroid may be felt pulsating strongly, the thyroid almost as an aneurism. The gland enlarges, one lobe—the right in the author's experience (six cases)—twice as often as the left; but ultimately the whole of the organ, in several months usually, after the increased pulsations have begun. In very rare cases no enlargement of the thyroid has occurred. Sometimes the goitre is the first symptom observed. It is elastic, rather soft, and has a distinct thrill like that of an aneurism. It never attains a very great size, reaches its maximum in a few days or weeks, and fluctuates greatly in its dimensions. During the exacerbations in the action of the heart it enlarges, and subsides correspondingly after the attack is over. After a time it becomes firmer, and remains uniform in size. This change is due to the fact that the variations in the volume of the gland are produced by the varying caliber of the vessels, and, when hyperplasia of the gland-elements occurs, the fluctuations in size are no longer possible. Very great changes in the thyroid may take place, due entirely to accidental causes. Thus it enlarges in pregnancy, and it may take on cystic and calcareous degeneration. Exophthalmus may be the first symptom, as in one of the author's cases, but usually this comes on after the goitre. It may begin in one eye, but it is very rarely confined to one, and usually one eye protrudes more than the other. It may not occur at all in a case otherwise well marked, but this is unusual. The degree of protrusion varies from a slight, staring expression to the actual dislocation of the eye on the cheek, and it increases during the paroxysms of active palpitation, and diminishes in the interval. A very important diagnostic point is the incoördination in the movements of the upper eyelid and of the ocular globe. If a patient be told to look at her feet, the upper lid, it will be seen, does not follow the movement of the globe. As this does not obtain in the exophthalmus from any other cause, and as it may be present early in the history of the case, it may be very important. The nutrition of the cornea may suffer, and conjunctivitis is an ordinary complication. More or less fever occurs during the course of this disease, and a very considerable subjective sense of heat is felt. The rise of temperature is from one to three degrees of Fahrenheit, and a considerable increase of sweat is observed. Pigment deposits and pityriasis versicolor have been observed by the author in some cases, and other trophic affections of the skin have been reported by Bulkley, of New York. Changes in the disposition are constantly observed. The subjects of this disease are nervous, apprehensive, irritable, and lachrymose. Vertigo, wakefulness, tremors, headache, impaired memory and power of application are often experienced. The appetite is usually poor, digestion feeble, vomiting readily occurs, and a more or less rapid decline in flesh and strength takes place. A marked degree of pallor is usually observed. The blood is anæmic, and amenorrhœa is present in most cases.

Course, Duration, and Termination.—Acute cases going through a full development and decline in a few months are very exceptional. It is an essentially chronic malady, and years are occupied in its varying phases. Recovery may ensue within six months, but usually it is not complete, and the symptoms develop again. The most important lesions occurring are dilatation of the cavities of the heart, and death as the ultimate result of the disturbances in the circulation. Tuberculosis is apt to supervene, and some cases are carried off by intercurrent inflammatory affections. A favorable termination may be looked for when the general health is good, the thyroid unchanged, except by simple hyperplasia, and the heart is sound.

Treatment.—The usual arterial sedatives possess but small value in the treatment of this disease. Good results have been obtained from belladonna and ergot. They should be administered for several months, and in full doses. The anæmia, which is so pronounced a symptom, requires iron. Traube achieved great success by a combination of quinine and iron. The author has had good effects from quinine, belladonna, and ergotin, in combination. Galvanization of the cervical sympathetic and the pneumogastric, by placing the anode under the ear and the cathode at the epigastrium, the author has found to be of the highest efficiency. While the current is passing, the action of the heart becomes less tumultuous, the protrusion of the eyes diminishes, and the thyroid shrinks somewhat. Besides the stabile application just indicated, labile applications should be made over the thyroid, and a weaker current should be applied to the eyes. While the galvanic applications are making, the remedies suggested may be used internally.

MYXŒDEMA.

Definition.—The term *myxœdema* is compounded of two Greek words—*μύξα*, *mucus*, and *οἴδημα*, *a swelling*—and is applied to a progressive disease, characterized by the occurrence of an apparent œdema, produced by the deposit in the textures of a mucoid substance, and by changes in the mental condition. The first account of it was given by Sir William Gull,* in a paper with the following title: "On a Cretinoid State, supervening after Adult Life in Women." The term *myxœdema* was proposed by Dr. William M. Ord, † who also gave the first correct, although by no means complete, account of the apparent œdema.

Causes.—The disease was at first supposed to occur in women only, and at the middle period of life, or about the climacteric. Dr. Morvan, ‡ who has made a collection of fifteen unpublished cases, reports one case as occurring in a man; Dr. Andrew Clark, of London, has met with several cases in men; and the author has now a perfectly well-marked case in a man of forty-two. The original statements in regard to the age must also be modified. A case has recently been observed in a girl of eleven, and in Morvan's collection the youngest was twenty-two. The conditions producing the disease are not well understood. Alcoholism, syphilis, metallic poisoning, have not been concerned in the cases thus far reported. Nine out of fourteen occurred in women at the climacteric period. Prolonged lactation has appeared to have a causative influence in a few cases, domestic worry has been alleged in others, and living in damp, unhealthy habitations has apparently produced the disease in a few instances.

Pathological Anatomy.—The essential condition of myxœdema is an overgrowth of the connective tissue, associated with a peculiar form of degeneration. The cement substance, containing more or less mucin in the normal state, is in this malady enormously increased; the fibrillæ undergo extensive hyperplasia and hypertrophic thickening, and the connective-tissue corpuscles multiply wherever there are connective-tissue elements—in the skin, mucous membranes, arterial tunics, glands, and nervous matter—there this peculiar change will be found to have taken place. The thyroid gland atrophies, so that its proper elements finally disappear, it may be, entirely. The overgrowth of the connective tissue causes an atrophic change in all the tissues encroached on, and in this fact we have an explanation of the various symptomatic derangements.

Symptoms.—The physiognomy of the subjects affected by myxœdema is very striking. The face is puffy, pallid, and earthy in hue; the lower eyelids are especially swollen by protuberant bladders, semi-transparent and œdematous-looking, so like that state in which they are in advanced renal disease that the first impression is the case is one of albuminuria; the lips are swollen, the alæ nasi thickened and protuberant, and the cheeks have a pinkish flush, terminating abruptly just below the orbit. There is no pitting of this apparently œdematous face; the skin has a rather doughy feel, is thick, resistant, and at the same time has a certain elasticity. The skin is generally dry, rough, and scaly, having a somewhat translucent aspect, and is without perspiration. The hands assume a peculiar shape, become square or "spade-like," as entitled by Gull, and the fingers are blunted, short, and somewhat clubbed.

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* "Transactions of the Clinical Society of London," vol. vii, p. 180.

† "On Myxœdema, a Term proposed to be applied to an Essential Condition in the 'Cretinoid' Affection occasionally observed in Middle-aged Women."—"Medico-Chirurgical Transactions," vol. lxi, p. 57.

‡ "Gazette Hebdomadaire," August and September, 1881.

The thyroid gland, in sharp contradistinction from the condition in cretins, is either much smaller than normal, or has entirely disappeared; but the connective tissue of the subclavian triangle is, on the other hand, tumefied and more or less elastic.

As the changes in the connective tissue include the neuroglia, it is not surprising that nervous derangements accompany the other symptoms. The expression of the subjects of this disease is heavy, stupid, and somewhat sad; the speech is slow and hesitating, if not incorrect; the voice monotonous, and the manner that of a person having an inactive, if not feeble, mind. The memory is impaired, the judgment uncertain, and the mental operations in general below the usual capacity. This depression in the mental corresponds to the enfeebled state of the motor and sensory functions. The muscular movements are slowly executed, more or less uncertain, without being actually incoordinate, and hence the handwriting, as well as the gait, become irregular, or awkward, without being otherwise perverted. There is a certain stiffness and clumsiness of the walk, without there being any actual loss of power, and more time is consumed in executing given movements than was usual in health. The same slowness and clumsiness are observable in all the mental operations. The response to a question is slow, partly in consequence of the tardy receptivity of the impression from without, and partly in consequence of the sluggish movements of the ideational centers.

The respiration is slow, the action of the heart rather depressed and comparatively feeble. The temperature of the body is habitually from a half to one degree below normal, and a subjective sense of chilliness is nearly constant. A feeling of fatigue is nearly always present, and exertion increases it. Active movements soon exhaust the strength, and breathlessness follows on moderate exertion. The nutrition of the body is poor, the appetite feeble, and the digestion languid. A peculiar and very persistent taste—bitter, sweet, or mawkish—is a frequent symptom. The hair has a dry and unhealthy look, and is apt to fall out to a less or greater extent, becoming very scanty at last. The nails are brittle, curved, and irregular in growth, and the teeth decay early. The skin being without its natural secretion, the kidneys are not opposed in function, and hence the amount of urine is increased. As the functions in general are sluggish, this increase of urinary water must be connected with the lessened activity of the sudoriparous glands. The change in the condition of the urine, at an early stage of the disease, consists entirely in the increased excretion of water, there being neither albumen nor sugar present. With the further progress of the mucoid changes in the kidney, and the encroachment of the connective tissue on the Malpighian tufts and tubules, the urine becomes albuminous.

The uterine functions do not appear to be affected by the progress of the disease. If the malady appears at the climacteric period, the disturbances belonging to this period are not necessarily related to the development of this affection.

Course, Duration, and Termination.—Myxœdema pursues a progressive course, and has apparently been little affected by remedies. It develops slowly, the appearance of œdema being secondary to the anæmia. As it develops there is a constantly increasing weakness. The mind, at first torpid, becomes a prey to hallucinations. The temper, at first amiable and slow to anger, becomes irritable. At or near the close of the malady stupor comes on, and the end may occur in coma. As the changes progress in the various organs concerned in assimilation, the nutrition fails, the muscular force is quickly expended, fixed attitudes are maintained with difficulty, and all exertion is finally accomplished with great difficulty. The blood becoming watery, and the urine albuminous, a true œdema finally comes on. The whole duration of an uncomplicated case is about six years. Intercurrent diseases may end life meanwhile. Death may occur from exhaustion, by uræmic poisoning, or by cerebral coma.

The prognosis, from our present stand-point, must be regarded as unfavorable. Nevertheless, increasing experience justifies the expression of some confidence in the good effects of remedies now utilized in the treatment.

Therapy.—Recognizable causes—as child-bearing, lactation, the affections incident to the climacteric period, bad hygiene, depressing moral emotions—should, as far as possible, be removed.

Remedies belonging to the group of nervous tonics—as arsenic, iron, massage, faradism, and a generous diet—have in many cases done great good, and in a few have effected cures. Pilocarpus and warm baths have been very useful in some instances, the effect of both being to promote the action of the sudoriparous glands. Ord reports that in three cases the disease almost wholly disappeared under the use of pilocarpus—ten to sixty drops of the fluid extract being given four times a day.

The author believes that the method of treatment known as central galvanization will prove to be an important aid to other measures. In a case now under observation, great improvement has followed the use of extract of ergot and arseniate of iron—two grains of the former, and one tenth of a grain of the latter, three times a day.

GENERAL OR CONSTITUTIONAL DISEASES.

ERUPTIVE FEVERS.

VARIOLA.

Definition.—*Variola* is an eruptive disease characterized by the presence of pustules, which make their appearance at the end of the third exacerbation of the initial fever, when the temperature declines, but this period of diminished fever or of apyrexia is followed by a secondary fever, or fever of maturation. *Small-pox*, or *pock*, is the name in common use, which was formerly employed in contradistinction to the *big pock*, or *syphilis*—the word "pock" meaning *pustule*.

Causes.—Small-pox prevails under all conditions of soil and climate, its distribution at the present time being regulated by the degree of protection afforded by vaccination.* It occurs at all ages, and even the *fœtus in utero* is attacked, and it may be so early as the fourth or fifth month of utero-gestation. Both sexes appear to be equally susceptible. Race exercises an influence which is quite decided—the dark races, negroes especially, possess a peculiar liability. During the actual existence of typhoid fever, scarlet fever, and measles, there is an immunity against the small-pox poison, and the susceptibility of individuals varies at different times. As a rule, those who have been attacked once possess complete protection against future seizures, but there are numerous exceptions. The author has met with examples of small-pox occurring twice and three times in the same individuals, and notwithstanding vaccination. The susceptibility to a new attack may be acquired in a few months, but usually not until many years have elapsed. Mild attacks are apparently less protective than severe ones against future recurrence of the disease. Small-pox is spread by a peculiar virus whose nature is unknown. It is true, minute organisms on which the toxic activity is supposed to depend have been found in vaccinia, and also in the pustules of variola, but their position, as accidental or causative, has not yet been made out. The transparent fluid of the pustules, before it becomes yellow and turbid, is most active, but the dried pustules are only less active. The morbific principle is not confined to the patient, but diffuses in the atmosphere about him, and extends a variable distance. Ventilation and large air-space dilute the poison ; hence a close room, with a number of persons, especially having small-pox, occupying it, concentrates the poison, making communication more certain. There is no period, from the initial fever to the final desquamation, at which the disease may not be communicated to the susceptible, but the stage of suppuration is the most virulent. All articles which have been about the person or bedding of the patient, especially those having a rough surface to which it may adhere, may retain the poison for a long time, and it may be conveyed from the patient, and from his bedding or clothing, to the clothing of another. The bodies of those dead of variola communicate the disease, probably until the virus is destroyed by putrefactive decomposition. The persistence in the activity of the poison and its power to resist external influences are very great. When preserved from the contact of air, it retains its activity for months and years. The spread of small-pox is affected by the immunity derived from attacks of the disease, but especially by the protective influence of vaccination. The exemption derived by the latter is less permanent than the former, and in many cases terminates after some years. It happens in this way that every few years a part of the population of civilized communities reacquire their susceptibility to the poison, and furnish the material for an epidemic.

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* "Traité de Climatologie Médicale," *op. cit.*, vol. iv, p. 370.

Pathological Anatomy.—The most important changes are those concerned in the formation of the pustules. The first step is the appearance of a small hyperæmic spot in the entire thickness of the derma, at and through a papilla. A swelling ensues in the part, especially in the outer layer of cells of the papilla, and pushing up the epidermis forms a papule. An exudation of a transparent fluid now takes place from the papillary layer, which, pushing aside the cells and the epidermis above, forms a vesicle. The cells are separated into groups, and not from each other, are compressed by the exudation, form an apparent network, in the meshes of which the lymph is contained.* While the upper cells of the papilla and the epidermis are engaged in the formation of the vesicle, the papillæ themselves are swollen by enlarged and tortuous vessels, and by an exudation of serum. A central depression—an umbilication—forms in the vesicle, which is perforated by a hair-follicle, or the duct of a sweat-gland. This is due to the fact that the epidermis is continuous with the hair-follicle, and the duct of the sweat-gland also, so that this portion of the vesicle can not rise—in fact, the accumulation takes place around it—whence it follows that a central depression must exist. Pocks without being so situated—not perforated by a hair-follicle or sweat-gland—also have this umbilication. Under these circumstances, we may adopt the explanation of Anspitz and Basch, who hold that this appearance is due simply to the more rapid swelling of the periphery of the pustule. When fully ripe the umbilication disappears, to reappear again in drying, owing to the more rapid desiccation of the center. In the case of confluent variola, the adjacent papilla may become inflamed, and partial necrobiosis occur, causing great destruction of tissue. There is nothing peculiar and distinctive in the hæmorrhagic form, this condition being due merely to the substitution of blood for serum. The hæmorrhage may be confined to the pustules, or may extend into the neighboring papilla, and, in the worst cases, the whole cutis and subcutaneous tissue may be infiltrated with blood. Pustules are formed on the mucous membrane, and simultaneously catarrhal, croupous, or diphtheritic inflammation takes place. The mucous membrane nearest the external skin, and most exposed, is most certainly and severely affected. The nose, tongue, tonsils, palate, and pharynx, and the orifice and internal portion of the Eustachian tube, are more or less infiltrated with pus; the tongue loses its epithelium to a considerable extent, and pustules extend down the trachea to the bifurcation, and also to a short distance down the œsophagus. The liver, spleen, kidneys, and heart are affected by granular and fatty degeneration, and in the hæmorrhagic form there are numerous hæmorrhages and ecchymoses throughout the body—in the serous and mucous membranes, and in most of the viscera.

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* "Untersuchungen zur Anatomie des Blatternprozesses," von Dr. H. Anspitz und Dr. S. Basch, in Virchow's "Archiv," Band xxviii, p. 337, *et seq.*

Symptoms.—The period intervening between the reception of the materies morbi and the outbreak of the malady is called the *period of incubation.* This is not a fixed period, although tolerably constant, the variations being due probably to the differences in susceptibility, to the action of the poison. The most usual period of incubation is from ten to thirteen days (Curschmann), which is the time generally agreed on by the authorities, but in some instances it has been as short as five and as long as fourteen days. During the stage of incubation we may suppose that the multiplication of the poison is taking place, but there are no objective nor subjective sensations indicative of the process until the *stage of invasion.* This stage sets in suddenly with a violent rigor, only comparable to that of an intermittent, or of pneumonia. Sometimes there are several chills or several hours of chilliness. Fever begins at once, and in a short time rises to 103° or 104° Fahr., at which it continues, there being a slight morning remission. The fever may rise higher after the first day, to 105°, even 106°, and continue at that rate until the period of eruption. The pulse is strong, full, and bounding, and ranges in adults from 100 to 140; in children to 160. The action of the heart is strong and heaving, there is some difficulty of breathing, often considerable dyspnœa is present, the carotids beat vehemently, the face is red, the eyes injected ; there are an intense headache and sleeplessness, or sleep is disturbed by frightful dreams. Appetite is entirely absent, thirst is incessant, and nausea and vomiting with constipation usually occur. There is present in all cases more or less pain in the back, but in the largest number this takes the form of agonizing suffering, the pain being at the same time acute, lancinating, shooting down through the hips and thighs into the lower limbs, and heavy, tensive, boring pain felt deeply in the spine. The pronounced backache is accompanied by the equally pronounced headache, which possesses similar characteristics. There may be some confusion of mind in the milder cases occurring toward evening, and in other cases active delirium, especially in subjects addicted to alcoholic excess. It was a dictum of Sydenham, revived by Trousseau, that the mildness and shortness of the stage of invasion furnished a guide to the character of the attack. "When the eruption makes its appearance at the end of the second day or the beginning of the third, it is necessarily confluent ; when it appears at the end of three and a half to four full days, or is postponed to the fifth, it is certainly discrete" (Jaccoud). Although there is a measure of truth in the former propositions, they are by no means exact. The author agrees with the dictum of Jaccoud. If the eruption appears after four full days of the preliminary fever, it is never confluent ; it is either discrete or coherent. Although definite conclusions as to the severity of the disease can not be drawn from the date of the appearance of the eruption, yet the severity of the symptoms during the stage of invasion does furnish a measure of the probable violence of the disease. Besides the regular phenomena belonging to the stage of invasion, there are certain inconstant symptoms which possess a high degree of importance. These are convulsions, delirium, and dyspnœa, which have been briefly referred to, and certain initial or prodromal rashes which have not thus far been discussed. Following the division of Curschmann, these rashes may be arranged in two groups, *erythematous* and *hæmorrhagic*. The erythematous form is general to the whole surface, and assumes either a scarlatinal or rubeolous appearance. The hæmorrhagic eruption consists of minute points of hæmorrhagic extravasation into the epidermis. When these are combined, the hæmorrhagic spots appear like petechiæ or an erythema. The favorite site for these initial rashes is the lower portion of the abdomen, the genitals and thighs forming a triangle which has been designated the triangle of Simon. "A brachial triangle" is formed of the rashes along the side of the trunk, extending into the axilla, the inner side of the arm, and over the pectoral muscles. The erythematous eruptions tend to spread over the whole surface of the body. These eruptions or rashes of the stage of invasion are by no means constant in their appearance; many cases and some epidemics are entirely free of them. In the last epidemic of small-pox, the author, then practicing at Cincinnati, saw a number of them, and it was common to hear reports of cases in societies of the coincident appearance and development of scarlet fever or measles and small-pox. They usually appear on the second day, but they may appear on the first or third. Their duration is short, the erythematous lasting from twelve to twenty-four hours, the hæmorrhagic a day or two or longer. The invasion stage of variola is sometimes diversified by the appearance of the hæmorrhagic condition or diathesis (*purpura variolosa*), and this is often confounded with the comparatively innocent hæmorrhagic rash. Purpura variolosa sets in in the usual way with severe rigor and pains in the head and back, very high fever, and great prostration. In from eighteen to thirty-six hours a very intense scarlatiniform eruption spreads all over the body except the face. Petechiæ and considerable patches of hæmorrhage appear in the skin and vary in size from mere points to an irregularly rounded figure about an inch in diameter, which remain discrete or apart on the extremities, and confluent on the abdomen, forming irregular masses. The face swells and is red; the conjunctivæ are injected, and the eyes, apparently sunken in their orbits, are surrounded by large, dark rings formed by the effusion of blood into the lids. The tongue is swollen and coated with a heavy, yellowish fur, and the pharynx, tonsils, and palate are covered with a membranous exudation, which emits a horrible odor. A severe cough, with watery and bloody expectoration, comes on, and there are nausea and vomiting, with bilious and bloody evacuations, and offensive bloody stools. The urine contains a large amount of albumin, which presently becomes bloody and thick. If pregnancy exist, miscarriage takes place, and the patient is carried off with the severe and uncontrollable hæmorrhage. The mind usually remains unaffected, though there may be delirium and coma, and death ensues somewhere from the third to the sixth day after the attack began. The body has a frightful appearance at and immediately subsequent to death; it turns black and is bloated, the features being horribly distorted and swollen. In such cases death appears before the eruption can develop, or it is so obscured as not to be recognizable.

Stage of Eruption.—The characteristic eruption makes its appearance at the end of the third exacerbation of the fever—usually on the evening of the third or the morning of the fourth day—and is seen first on the forehead, about the eyes and mouth, on the hairy scalp, and then extends to the body and the extremities. The eruption at first consists of a red point, effaced by pressure, slightly elevated, somewhat hard, and rolling under the finger like a small shot in the skin. The manner of distribution of the eruption varies somewhat. There are four forms in which the eruption may be arranged: the *discrete*, or each pustule separate and distinct; the *corymbic*, or placed in clusters or patches; the *coherent*, in which the individual pustules come in contact; and the *confluent*, in which the pustules unite or flow together without a line of division between them. In the ordinary typical case, the eruption is completed and no new pustules make their appearance after twenty-four to thirty-six hours. They tend to locate about the hair-follicles, the orifices of the sebaceous and of the sudoriparous glands. On the second day of the eruption, and the fifth day, including the initial stage, the red point is enlarged and elevated into a papule. On the third day of the eruption the papules are transformed into vesicles filled with a transparent, serous fluid; the vesicles increase in size during the next day or day and a half, and on the fifth day of the eruption, and the eighth day of the disease, the serous fluid of the vesicle becomes milky and presently purulent. When the vesicle is fully developed, a central depression or umbilicus appears, and at the bottom is seen a hair-follicle, or duct of a sweat or sebaceous gland, but many distinctly umbilicated are not around a hair-follicle or gland-duct, showing that this appearance is in part due to the more rapid development of the peripheral portion, as suggested by Anspitz and Basch. If the summit of the vesicle which now appears milky be punctured, a drop only of fluid will escape, because of the cellular arrangement of the body of the pustule. While the appearance of the eruption does not indicate suppuration in all the forms, except the confluent, until the eighth day, the fever of suppuration really begins on the seventh. In the confluent and the extensive coherent forms, the fever of suppuration may set in on the sixth day of the period of eruption. There may be, therefore, considerable variation in the duration of the stage of eruption.

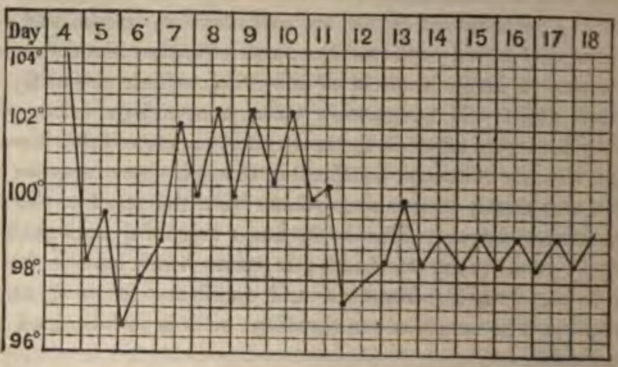

FIG. 50.—Range of Temperature in Discrete Variola.

In the discrete form, a marked change takes place in the condition of the patient when the eruption appears. The intolerable headache and backache subside or disappear entirely, the fever abruptly falls to normal, even slightly below it, the nausea ceases, and the patient within a few hours passes from a condition of great suffering to one of comfort. It is only in cases of varioloid, or of variola, with few pustules, that the defervescence is so sudden. In the more severe discrete cases, or coherent, the decline of temperature, although considerable, does not reach the normal, and occupies a day or two of a remittent type, with considerable exacerbations. In the confluent form there is a mere abortive attempt at decline of temperature without much change. The pulse falls correspondingly to the decline of fever. An eruption appears on the mucous surfaces simultaneously with that on the skin: on the conjunctiva, pharynx, larynx, and trachea, and causing lachrymation, photophobia, difficulty in swallowing, and cough. In confluent cases the eruption may extend to the bronchi, to the intestine, urethra, and vagina. Very serious results may be derived from the pustules in these situations. Besides the symptoms above mentioned, there may be a violent conjunctivitis, ulceration of the cornea and staphyloma, with intense photophobia; constant flow of saliva, and dysphagia; toneless voice, croupy cough, and suffocative attacks; dysenteric discharges; painful urination; and a sense of burning in the vulva and vagina. In addition to these constant symptoms there are others that may be regarded as accidental. If stupor and delirium appear during the stage of eruption, these symptoms are of evil augury. If merely due to habits of alcoholic excess, they are less serious than if they arise spontaneously under an increased mobility or instability of

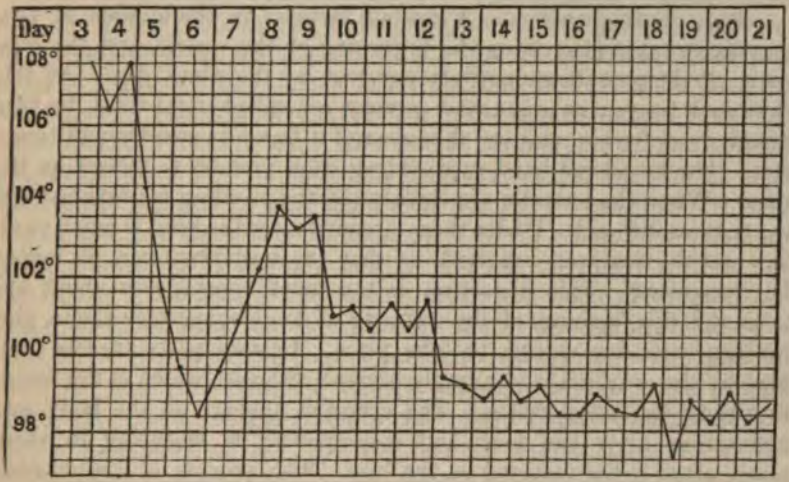

FIG. 51.—Range of Temperature in Coherent Variola.

the nerve-centers, and changes in the composition of the blood. If there be maniacal delirium, with suicidal tendency, the result is usually death about the beginning of the stage of suppuration (Jaccoud).

Stage of Suppuration.—A gradual increase in the number of pus-corpuscles takes place from the beginning of the vesicle, and the contents of the pustule are entirely purulent by the ninth day. At this time each pustule enlarges, and assumes a hemispherical form, its base broader and darker, and the skin around it becomes swollen and tumefied. The broad red band surrounding each pustule is known as the halo. When the pustules are thickly set, the swelling is universal and the redness diffused. Under these circumstances the head is much swollen, and the features distorted, so that the individual can no longer be recognized. This distortion is the more conspicuous about the eyes and lips, because of the quantity of loose connective tissue, permitting extensive œdema to take place. Certain parts are less troubled by the eruption, and notably Simon's triangle, which is the favorite site of the initial rashes. The eruption appears on the body and extremities after the face, and consequently is maturing in these places after it has matured on the face. The process of suppuration in the pustules is accompanied by a symptomatic fever. A chill, or a succession of chills, mark its onset in some subjects, but this remark is true of those cases only in which the appearance of the eruption was coincident with a defervescence of the fever, or at least with a considerable decline. When the fever has persisted from the beginning, it is increased by the suppuration, and assumes a somewhat different type, becomes remittent, the daily variations being as much as two degrees. The range of temperature and the pulse-rate, as well as the various kinds of disturbance accompanying the fever, are greatly influenced by the extent of the suppuration. The temperature will rise to 104°, 105°, or 106° Fahr., and the pulse to 100, 120, 140, or higher. With the development of the secondary fever, there will appear all of the distressing sensations which marked the initial stage. The headache and backache again become severe, the whole surface of the body is full of the pain and irritation of the suppurating sores, there are great restlessness and wakefulness, and an active or low-muttering delirium comes on. Frequently the delirium is maniacal, and the patients difficult to restrain: they jump out of the bed, or out of the window, or escape into the streets. In children, the heat and burning of the face are so great that they will scratch the parts, covering their hands and the bedclothes with blood, and greatly increasing the local inflammation.

The drying of the pustules begins about the eleventh day—rarely earlier, more frequently later—and in the order which the eruption followed. The drying begins before the disappearance of the fever of suppuration, for, when the face-pustules have completed their evolution, those of the extremities are just suppurating. When the desiccation begins, a honey-like exudation is poured out on the surface of the pustules, which, drying, forms an adherent coating. The contents of the pustules also desiccating, a brownish scab results. Before desiccation has taken place in the pustules on the posterior portions of the body, the matter which they contain is pressed out on the bedding and clothing of the patient, and, decomposing, a peculiar odor results, which to many persons has something distinctive, even diagnostic, about it. Owing to the thickness and hardness of the epidermis, the pustules on the hands and feet have a peculiar form and dry earlier, but are slower to separate. As the pustules dry, the redness and swelling of the skin subside, and the face begins to assume something of its natural appearance, albeit somewhat roughened, reddened, and disfigured by the disease. Although the whole body is marked by cicatrices, the face is peculiarly disfigured. The pustules involving the true skin, and closely placed, extensive losses of substance may occur, especially about the nose. Ulcers penetrating the cornea, protrusion of the lens, and various opacities, result. A depressed and radiated cicatrix, becoming whiter than the surrounding skin, is left at the site of every variola pustule. As the crusts are detaching, there is often an intolerable itching, and injury is done by children who increase the area of inflammation by the violence of the scratching. Erysipelas may occur and furuncles form during the progress of the dermatitis. The hair usually falls out, and the nails sometimes drop off.

CONFLUENT VARIOLA.

The description above given applies to the ordinary cases of small-pox : to the discrete, the corymbic, and the coherent. There are some peculiarities of other forms which require particular consideration. The approach of the confluent form is announced by the greater violence of the initial or invasion stage, and by the earlier appearance of the eruption. When the eruption appears it spreads over the body quickly, and indeed, in some cases, it seems to be on the face, body, and extremities simultaneously. At once the papules approximate, and their entire formation is prevented by the closeness of arrangement, so that large numbers coalescing form immense vesicles filled with sero-pus. While the face and features are hidden under huge bullæ of pus, the pustules on the rest of the body may be merely coherent. The mucous membrane is attacked with similar violence ; the pustules flow together, and diphtheritic exudations spread over the fauces, pharynx, nares, and Eustachian tubes. The tongue is greatly swollen, and protrudes from the mouth. Pustules form in the larynx, the cartilages are invaded, abscesses develop, and œdema of the glottis ensues. The parotid and sublingual glands swell enormously. The cornea is opened by ulcerations, and staphyloma results. Erysipelas, phlegmonous inflammation, and extensive suppuration may occur in those parts where the eruption is most confluent, and even gangrene results in extreme cases. The systemic state, as might be expected, is quite in harmony with the condition of the skin and mucous membrane. During the initial or invasion stage, the temperature reaches the highest point human temperature ever attains, and declines but little, sometimes not at all, and always slowly when the eruption appears, continuing at 104° to 105°. The stomach is very unsettled, and vomiting is incessant, scarcely anything being retained. The urine is scanty, and loaded with albumin. If the patient pass through the dangers of the invasion fever, the eruption and suppuration stages, there will occur in the stage of desiccation extensive losses of substance of the skin of the face, eyelids, and eyes, and of the scalp, so that very great deformity, with baldness, will result.

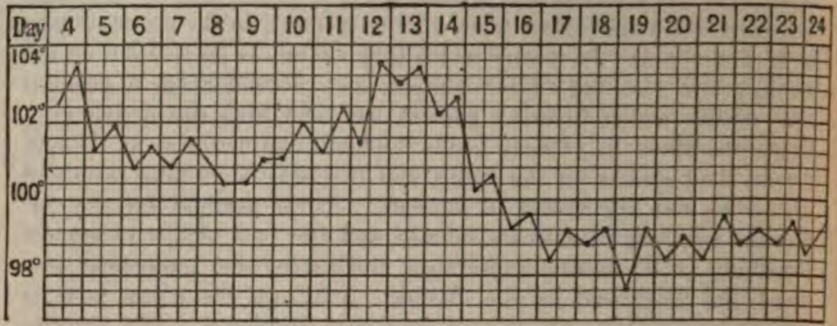

FIG. 52.—Range of Temperature in Mild Confluent Variola.

HÆMORRHAGIC VARIOLA.

It is important not to confound hæmorrhage into the pustules, or *hæmorrhagic variola*, with *purpura variolosa*, which is the hæmorrhagic diathesis superadded to the phenomena of variola. Again, the hæmorrhagic rash of the invasion stage—merely petechiæ—is quite distinct from the other forms. There may occur, with hæmorrhage into the vesicles, extravasations of blood into the adjacent parts. Only a portion of the eruption may be affected by hæmorrhage into the pustules, or it may be general over the body. Blood may escape into the papules, or not until the stage of vesicles is reached, but the most usual condition is for the hæmorrhage to occur when the pustule is well umbilicated. It usually takes place by degrees, beginning on the lower extremities. The mucous membranes of the mouth and throat are marked by extensive ecchymoses, and diphtheritic exudations spread over the tonsils, palate, and pharynx. With these troubles are associated a spongy state of the gums, and hæmorrhages from the nose, gums, kidneys, uterus, and, if pregnancy exists, abortion followed by metrorrhagia. The general condition corresponds. The profound alteration in the composition of the blood manifest under these circumstances is accompanied by very great prostration of the vital forces. But there are great differences in the gravity of these cases, as there are in the extent of the hæmorrhagic extravasations; in some epidemics the hæmorrhagic pustules are not numerous, and the general condition not unfavorable.

VARIOLOID.

Varioloid is a form of variola modified by previous vaccination, by a former attack of variola, or by some special insusceptibility to the action of the poison. It is, however, at the present time, almost wholly the influence of vaccination, which so modifies small-pox as to cause it to take the mild form or varioloid. The protective influence of vaccination, or of an attack of the real disease, is at first complete, but the longer the time which elapses from the date of the vaccination the less protective its influence; but in many persons, it is true, this protection continues throughout life. It is a peculiarity of varioloid that it presents numerous points of departure from the typical course of variola. Thus the stage of invasion may be one or two days, or three or four; and the temperature declines very abruptly at or just after the appearance of the eruption, and descends to or below normal, and it remains at normal until the stage of suppuration, when it assumes a transient rise of not more than one or two degrees. The initial or invasion rashes of the erythematous variety belong to varioloid, and not to variola, and the more decided the rash the less abundant the pustules. Great diversityand difference, as compared with variola, exist in respect to the manner of development and characteristics of the varioloid eruption. It does not always appear first on the face, but on the chest, abdomen, or extremities; it may all appear simultaneously over the body, or there may be a very slow eruption of the pustules. While the structure of the varioloid pustule does not differ from that of variola in respect to development, there are remarkable variations. The eruption, although it may apparently be as complete as variola, never goes through the development of the latter, and they abort at different stages. They may not proceed beyond mere papules; they may develop into vesicles and then dry up; they may become pustules, surrounded by a red areola, but the surrounding skin is not swollen, and from the fifth to the seventh day of the eruption desiccation occurs. The pustules containing a sero-purulent fluid dry up without discharging, and, although an hyperæmic spot remains for a short time, no scar is left. The eruption on the mucous membrane is usually slight, and produces but little disturbance.

Course, Duration, and Termination.—The discrete, corymbic, and coherent forms are severe, according to the extent and number of the pustules, and pursue a course of great uniformity. The most formidable of all the varieties is that known as *purpura variolosa*—the hæmorrhagic condition or diathesis superadded to small-pox. Death takes place in this form before the characteristic eruption appears or has time to develop; rarely do any cases live beyond the sixth day of the disease. The confluent form, although largely fatal, is not invariably so. The termination is usually by pneumonia, pleurisy, or pericarditis, especially the last two. When recovery ensues, the convalescence is tedious, and interrupted by various complications, especially abscesses of the skin. Very often the termination is by pyæmia. The hæmorrhagic pustular form is characterized by great intensity of the lumbar pain, and by remarkably low temperature, which may persist throughout. On the other hand, the temperature through the initial stage and subsequently may be very high. This form is more protracted than purpura variolosa, and almost as fatal. The mortality, however, is very much affected by the number of pustules into which hæmorrhagic extravasation has taken place. The author has in one epidemic seen at least four cases recover out of six of the hæmorrhagic form, but the pustules of the face were chiefly affected. The course of small-pox is modified by various complications. Numerous points of inflammation exist throughout the brain and spinal cord in some cases. Serious complications on the part of the eye have already been mentioned, consisting of ulcerations of the cornea, panophthalmitis, hæmorrhage into the retina, etc. Chronic otitis, caries of the bones, and permanent loss of hearing result, and the voice is hurt by chronic inflammation of the larynx. The mortality is much affected by the age of those attacked: at the extremes of life, notably in infancy, the mortality is greater. In women, owing to the accidents growing out of pregnancy, the mortality is greater than in men. Alcoholic excess greatly increases the danger. All those circumstances lessening the vital power of individuals impair the power of resistance to the disease. The more extensive the eruption, as has been stated, the greater the danger. Next to the extent of the skin affection, as a measure of prognosis, stands the pustulation of the mucous membrane. Diphtheritic affections of the throat and inflammation of the larynx are very dangerous complications. The duration of any case depends on the form, extent of the eruption, the complications, etc. An ordinary case of discrete variola will not run its course under five or six weeks.

Treatment.—We postpone vaccination, a means of prophylaxis, for separate consideration. We possess no means of treatment to modify the course or shorten the duration of small-pox. All specifics may be dismissed with the assertion that they have, one by one, proved worth-less, from sarracenia to zylol. The treatment is therefore symptomatic. Assertions as to the value of special remedies, or plans of treatment, must be received with caution, since the almost universal practice of vaccination modifies the behavior of cases—effects which may be readily mistaken for the influence of the medication employed. When the case is one of varioloid, but little treatment is necessary. In the confluent form, treatment is as little important, because without effect. During the stage of invasion, the high temperature and the cerebral disturbance are the points to which we direct attention. To allay restlessness, delirium, and fever, bromide of potassium and chloral are the most efficient remedies. If the headache and backache are very intense, the hypodermatic injection of morphine should be practiced occasionally. The bromide, some believe, has the power to modify the eruption. When the secondary fever develops, the best remedies are quinine in five-grain doses, and bromide of potassium to allay cerebral excitement. So common is it for the delirium to assume a maniacal character that the utmost care is necessary to prevent accidents. Chloral is not advised to be given at this period by the stomach, because of its highly irritant effect on the fauces, but it may be given by the rectum. Morphine, or opium in some form, will be indispensable to relieve the painful sensations experienced by the patient. Depression of the powers of life will be best antagonized by the free use of carbonate of ammonia and alcoholic stimulants. From the beginning, proper aliment is necessary. Milk, eggs, animal broths, oysters, and beef-juice, should be given regularly from the beginning, every three hours. Ice is always grateful, and should be allowed freely. When there are many pustules in the mouth, ice should be held in the mouth as much as possible, and ice will best serve to allay nausea. If there is much vomiting, the hypodermatic injection of morphine is the most efficient remedy to arrest it. An ice-bag to the head and to the spine will afford much relief to the pain. For the eruption on the face numberless expedients have been resorted to, with a view to prevent pitting. The French employ, and, as they think, advantageously, a mercurial plaster. It is probable that a mask of some unctuous material, thoroughly applied to exclude the air, has a beneficial effect. The author has used with apparent advantage the glycerite of starch, freely applied by a large brush several times a day. As the papules are about to develop into vesicles, the tincture of iodine should be painted over them thoroughly. There are good reports from this practice. Of all the local applications, there is nothing so serviceable, according to Curschmann, as water-dressings to the face and hands. Cold compresses are kept constantly applied. They not only give great relief to the local heat and burning, but diminish the swelling of the skin. If cold is not pleasant, warm applications may be used instead. For the mouth-eruption, solution of chlorate of potassa, and, if there is much fetor, of carbolic acid, is useful. Astringents may also be used with advantage—such as fluid extract of hydrastis, of eucalyptus, and subsulphate of iron. When the crusts are falling off, warm baths assist in detaching them, and also allay the troublesome itching. Inunctions of lard, of suet, of vaseline, after the warm bath, are more effective. All the excreta of the patient should be at once disinfected by carbolic acid, sulphate of iron, iodine, etc. The air of the apartment should be also disinfected by the vapor of iodine, or by sulphurous acid, and the halls communicating with the room not less so. All articles about the patient should be destroyed, and the apartment renewed in all respects.

VACCINIA AND VACCINATION.

Vaccinia, or *cow-pox*, is a natural disease occurring in the cow and horse, and possibly some other animals. It is a vesicular disease, the eruption limited to the udder and teats, and occurs sporadically or as an epizoötic. It seems to be peculiar to milch-cows, and is conveyed to others by the hands of milkers. It is the young cows who are chiefly affected, and the course of it is essentially the same, whether it arises spontaneously or is propagated by inoculation. In the natural disease the period of incubation is usually three or four days, but it may continue from five to eight. The udder swells, becomes hot and tender, and hard papules, the size of a pea, appear at the base of or on the teat. When the disease occurs by inoculation, if there be a crack or an abrasion of the skin, a papule may develop as early as the fifth day, but, if the skin be unbroken, not until the eighth or ninth day. In three or four days after their first appearance, the papule has acquired a distinctly vesicular character, and a central pit or depression is then to be seen. In four days more, or in about eight days from the first manifestation of the papule, the formation is complete. They vary in number from two or three to twenty or more, and their usual size is about that of a dime. Their shape is somewhat influenced by their position: on the teats they are oval; at the base of the teat round; but both forms may appear on the udder, and on the teats they may be coherent, even confluent. Their color varies somewhat, but they usually have a shiny, glistening, metallic luster of the margin, with a slate-colored center. They are surrounded by a narrow areola, pale-rose or damask-colored, and a band of induration. The color and tints of the vesicle and of the areola differ somewhat, according to color and texture of the skin. When the development is completed, at the end of eleven days, the lymph is abundant; the central depression disappears, and instead there is a conoidal elevation. If it now burst or is opened, a quantity of a straw-colored or amber-colored lymph flows out; but, if rupture does not take place, the lymph becomes turbid and purulent, and by the fourteenth day a crust of a brownish-black, or rather mahogany, color has formed, the areola and the marginal band of induration subsiding. The crusts shrink, dry, and fall off about the twenty-third day. The cicatrix is smooth, oval, or circular, according to the shape of the vesicle, and whitish in color. When the vesicles are handled, and ruptured as in milking, there will be seen large black scabs adherent at some points, and a raw, bleeding surface at others, while here and there appears a properly formed vesicle. Examination of the structure of the vesicle demonstrates a number of partitions, and the lymph contained in the spaces formed by them—an arrangement just like that of the small-pox vesicle. The vaccine disease may be produced by inoculation with lymph taken from other cows suffering with the disease; with the lymph of horse-pox, which is identical with the cow-pox; with humanized lymph, or retro-vaccination; and by the matter of small-pox, or variolation. The latter process has given origin to a good deal of controversy, owing to the difficulty of inoculating cows with the matter of variola, but it has been accomplished a number of times, the results being in all respects the same as ordinary vaccinia—so that the vaccine disease, as Jenner originally maintained, is variola, modified by transmission through the system of the cow.

Vaccination.—It would be a misapplication of space to discuss the value of vaccination as a means of saving men from the greatest scourge of modern times. Shall humanized, Jennerian lymph, or bovine virus be used to vaccinate? The following facts seem conclusive in favor of the latter: The carelessness in selecting and storing the humanized lymph and the vast numbers of transmissions have impaired the quality of the product, and, although, so far as the development is concerned, it still conforms to the original type, its protective influence seems less. Again, owing to carelessness in collecting the lymph, the syphilitic virus has been inoculated with vaccine. Much prejudice has been excited against humanized lymph, and hence any unavoidable accident occurring from its use would be referred to a supposed impurity. For these reasons bovine lymph is preferable. The objections to the latter are, that it is less certain, and that its action is violent, a good deal of constitutional disturbance being caused by it. The lymph should be preserved on quills, or ivory points; and, if transported a long distance, in hermetically sealed tubes. It may be mixed with glycerine when intended to be kept in sealed tubes some time. When vaccination is performed with humanized lymph, it is preferable to use that of the fresh vesicle on the seventh or eighth day—or "arm-to-arm vaccination." The author has used successfully a number of times lymph that had been transported from Germany. The lymph is obtained from the vesicle of the seventh or eighth day, by carrying an incision around the outer border of the vesicle so as to open the several chambers of which it is composed, care being taken not to cut or injure the skin. With a fine pipette the lymph may now be withdrawn, and mixed with two parts of glycerine and two of distilled water, and preserved in capillary tubes, sealed hermetically with sealing-wax. The utmost care should be exercised in the selection of the children furnishing the lymph, and in the stock from which the virus is derived. In practicing vaccination, the skin should be rapidly and carefully scraped until the true skin is reached, and it is ready to bleed. The lymph may now be brushed over this surface with a camel's-hair brush. Another mode is to make three or four horizontal and transverse cuts about four lines long, or to insert the virus on the point of a knife by a single puncture. A little blood, but not much bleeding, should be caused by the cuts or punctures. Three or four points should be selected on the arm or leg for inserting the virus, and far enough apart so that the areola—certainly the vesicles—can not coalesce. If the vaccination "takes," a papule makes its appearance on the third day at the site of the puncture or incision; on the sixth day a vesicle has formed, of a bluish-white color, having a raised border and a central depression; on the eighth day it is fully formed, distended with lymph, and a reddish areola surrounds it, which widens to two inches or more, and there is very considerable induration of the skin and subcutaneous areolar tissue. The areola begins to fade on the tenth day, and the contents of the vesicle become turbid, yellowish, and thick, begin to dry, and by the fourteenth day a brown, mahogany scab or crust has formed, but is not detached until about the twenty-third day. A genuine crust is circular, has a rounded and elevated border, a central cup or depression, and it has a dark-brown or mahogany color. The cicatrix left is circular, depressed, radiated and foveated, and is usually permanent, becoming after a time paler and whiter than the surrounding integument. More or less constitutional disturbance attends vaccination in children with a mobile nervous system: fever, when the vesicle is at its maximum; restlessness at night, etc. An eruption of roseola may take place, or a papular eruption—a lichen—may appear. In scrofulous children an eczema may be produced from the irritation caused by the development of the vesicle, or an otorrhœa may follow, etc. The lymph is usually held responsible for such accidents, but in strumous subjects the slightest wound may be followed by the same cutaneous troubles. As the protection is for a period which varies in different individuals, and, although for the whole life in most subjects when properly done, expires in others in a few years, it is necessary to repeat it at certain periods. Revaccination, practiced now in the great Continental armies, has had a remarkable influence in checking small-pox, and, as these statistics are on an enormous scale and are accurate, the lesson taught us by them ought to be heeded. When there is some special exposure to contagion, vaccination should be practiced; but as a rule, and entirely irrespective of contagion, revaccination should be done about the fifth year, after the second dentition, and at puberty. If properly done at these times, further vaccination will be unnecessary.

VARICELLA.

Definition.—*Varicella* is a febrile affection, characterized by the appearance of a vesicular eruption with the first elevation of temperature, the vesicles drying up and falling off in from three to five days, the elevation of the temperature ceasing at the same time. It is known in common language as *chicken-pox.*

Causes.—That it is an independent, specific affection, propagated by some peculiar poison, is now generally admitted. Its identity with varioloid has been and is still maintained by some authorities, but on insufficient grounds. It is a disease of childhood; and rarely attacks any one above ten years of age. It occurs both sporadically and as an epidemic. The mode of communication is unknown, and, although contagious, is not actively so.

Pathological Anatomy.—The eruption is both discrete and corymbic —vesicles occur singly and in groups, and they vary in size from a pin's-head to a pea, reaching sometimes the size of a silver dime. They may be few in number, from ten to thirty, or they may be numerous, reaching one thousand. They consist of perfectly transparent vesicles, containing a clear, watery, sometimes yellowish fluid, faintly alkaline in reaction. They form on a spot which is slightly hyperæmic, and are surrounded by a faint areola, which is, however, often absent. They continue at their maximum not longer than a day, when they begin to be flaccid, dry in the center, and form a small, yellow, or brownish crust, which falls off in two or three days, leaving a faint reddish spot which disappears entirely in a few days, and sometimes a cicatrix, which, however, is shallow and very rarely permanent.

Symptoms.—The eruption of the vesicles is the first symptom to attract attention, for there is no fever of invasion, and no prodromes that have been accurately studied. With the appearance of the eruption, a rise of temperature begins, but it is not often the case that the temperature rises high enough to be a subject for solicitude, the thermometer marking one, two, rarely three degrees above normal. The eruption first appears on the trunk, and then extends quickly to the extremities. The hairy scalp usually contains a number. At first a spot of roseola appears, and on this is quickly projected a vesicle. Between the first crop of vesicles, on the next day, are seen a number of roseola-spots, and on these other vesicles make their appearance. But few appear on the face, and those chiefly on the forehead. The disease reaches its maximum on the second day and then declines, the fever disappearing, the vesicles drying up and dropping off. The vesicles also appear on the mucous membrane of the mouth and on the genitals. The general symptoms are trivial. With the fever there are thirst, anorexia, and constipation. Sleep is disturbed, and much itching is complained of, especially in the scalp. The eyes are apt to be irritable, and it occasionally happens that vesicles appear on the conjunctiva, but the popular notion that chicken-pox is hurtful to the eyes is unfounded.

Treatment.—There is nothing to be done but await the termination of the case by the natural mode.

MEASLES—RUBEOLA.

Definition.—*Measles* is an eruptive fever, with catarrhal symptoms referable to the broncho-pulmonary mucous membrane, self-limited, and terminating in about two weeks.

Causes.—According to Lombard, measles appears in all parts of the globe, but is much less severe in the tropics and in extreme northern countries. It is a contagious disease, which may be communicated not only by immediate contact with the sick, but the morbific principle adheres to fomites, to articles of clothing, etc., by which it may be conveyed long distances, and by means of the healthy. It has been communicated by inoculation. The nasal mucus seems rich in the morbific principle. Measles prevails widely as an epidemic, and it occurs also in the sporadic form. Susceptibility to it is not the same in all individuals. Infants at the breast are not liable. The two sexes are affected with equal frequency. During an epidemic, not all exposed to the epidemic influence have the disease. One attack, as a rule, gives exemption from future attacks; but to this dictum there are numerous exceptions. It is a disease of childhood especially, although infrequent in infants at the breast, and a few cases have been reported in which measles existed at birth. The atmospherical conditions which favor the production of bronchial attacks promote the epidemics of measles, which are therefore more numerous and severe in the fall, winter, and spring. The period of the disease when the contagion is most active is probably when the eruption is at its maximum; but the contagious principle is present from the beginning to the end of symptoms.

Pathological Anatomy.—The eruption of measles is in dark-red, sometimes rose-colored, spots, sharply defined, about the size of a pin-head to three pin-heads, disappearing on pressure, and immediately recurring when the pressure is removed. These spots have a lenticular shape, are usually discrete, and separated by tracts of normal skin, but may be coherent, forming an extended area of diffused redness, with punctations of deeper red, while the intervening skin is untouched. The spots are slightly raised above the general surface, and each spot may be surmounted with a very minute papule; but this papule is not always present. The eruption of measles, with or without a papule, makes the skin rough. The spots appear on all parts of the body, but more on the face and trunk than on the extremities; and they are more apt to cohere on the face and to be more abundant in this situation also, and of a brighter color. The exanthem appears first on the face, then on the neck, throat, upper part of the chest, and abdomen. It may develop fully on the face and continue there unchanged for a day or two before appearing elsewhere. The duration of the eruption at its maximum of development is not more than a half-day or a day, when retrocession goes on rapidly, beginning usually in the evening or at night, where the exanthem first came out, and in twenty-four hours the skin is pale. As the retrocession is going on, an exacerbation may occur, when the spots will appear again, almost to their original development; but this is exceptional, and, if it happen, fading will soon (in a few hours) go on again. Some color remains for a few days at the site of the eruption—a brownish or yellowish stain—and, in the case of hæmorrhagic extravasation, which may take place in the skin during the height of the eruption, the petechial spots pass through the ordinary changes. More or less exfoliation takes place in the form of furfuraceous scales, and only from the spots; large patches, like those of scarlet fever, are not known in measles. The mucous membrane is affected, as well as the skin, but in a different form. An intense hyperæmia of the nares, pharynx, palate, larynx, and conjunctiva, comes on with the initial stage. To this state of hyperæmia are superadded dark-red spots, appearing with and corresponding to the skin exanthem, although not resembling it very closely. Minute papules are also seen to develop, but not in connection with the red spots. Retrocession of the mucous-membrane exanthem occurs a little earlier than that on the skin. In the measles of the war of the rebellion, intestinal changes were constantly observed, and consisted of enlargement of the solitary glands, more or less thickening of the patches of Peyer, and swelling of the mesenteric glands. The spleen was always enlarged by increase of the splenic pulp, and the kidneys were intensely hyperæmic, the urine containing albumen. The blood was thin, the fibrin slight in quantity and feebly coagulable, the red corpuscles diminished and the white in excess.

Symptoms—Invasion Stage.—The onset of the disease is announced by a feeling of weariness, muscular soreness, headache and backache, and a succession of irregular chills, the temperature then rising to 100° or 101° Fahr. These symptoms, which mark the beginning of the prodromal or invasion stage, succeed to the incubation stage. From the period of exposure to the appearance of the eruption there are fourteen days, according to the most accurate observations. As four of these are occupied by the invasion stage, the period of incubation must be fixed at ten days, or from nine to eleven days. During the incubation period there is no recognizable departure from the normal, and the symptoms of the invasion stage come on rather abruptly. Together with the symptoms above mentioned as indicating the approach of measles, there is an intense nasal, pharyngeal, and laryngeal catarrh, which usually appears on the first, but may be postponed to the second day. The fever rises to 102°, where it usually remains for the first day or two, and its intensity furnishes a measure of the severity of the attack. On the second or third day—usually the second—a remarkable remission takes place, the temperature descending to normal or nearly so. On the evening of the third or the morning of the fourth day the fever rises again to the original height. With this decline in temperature, there ensues an improvement in the general condition: the headache ceases and the general discomfort lessens; but the catarrhal condition does not moderate; the nasal mucous membrane swells; breathing through the nose is difficult; there are frequent paroxysms of sneezing, and presently an abundant secretion of mucus is poured out from the membrane. The eyes are swollen, the conjunctivæ injected, the lids œdematous, and hot, scalding tears flow over the cheeks. During this time epistaxis is frequent, especially in children. By the third day the catarrh reaches the larynx, and then the voice becomes hoarse and husky, the cough harsh, resounding, metallic, stridulous. At first there is no expectoration, and only sibilant *râles*, but more or less præcordial oppression and anxiety are felt.

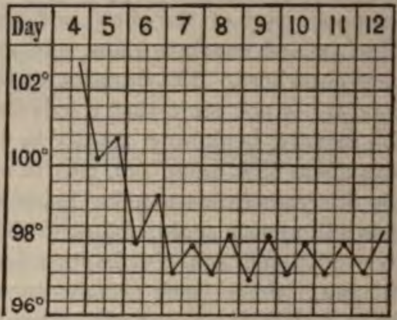

Fig. 53.—Range of Temperature in Uncomplicated Measles.

Eruption Stage.—The characteristic eruption of measles makes its appearance on the fourth day, and is rarely postponed to the fifth. In the milder cases the eruption appears on the morning of the fourth day; in the severer cases, in the after part of the same day; and it is seen first on the face, forehead, chin, and cheeks, spreading thence often, after an interval, over the body and extremities. The fever attains its maximum on the appearance of the eruption, or on the fifth day, or there may be remissions—the maximum on the evening of the fourth, and a remission on the morning of the fifth. The color of the spots is deepest when the temperature is highest. The condition of the mucous membrane continues the same, but the cough soon becomes easier because of the abundant secretion of mucus, soon assuming a muco-purulent character. Complications may arise at this point; considerable bronchitis may develop; diarrhœa comes on; albumen (usually a trace) appears in the urine. These symptoms were usual and constant at this period of army measles. About the seventh to the ninth day the eruption on the face begins to pale, and the turgescence and redness of the visage lessen. With the retrocession of the eruption the temperature declines somewhat, and the normal is reached in a day or two. The defervescence may be sudden and without interruption, the normal being reached in a day, or it may be gradual and varied by exacerbations and remissions. The slight desquamation that takes place is soon completed. Convalescence may be retarded by an irritable state of the intestinal canal.

Course, Complications, and Anomalies.—The course and behavior of measles are much affected by the character of the epidemic influence, by the susceptibility of the individual and the hygienic surroundings. As it prevails in armies, measles comes to be a formidable disease, comparable only to typhoid; sporadically, under favorable conditions, it is of very minor importance. In some epidemics many of the cases are very mild—cases of measles without the catarrh; other cases, in which the catarrh and other symptoms are present, but the eruption is absent. On the other hand, some epidemics are characterized by the severity of the cases. Thus, in some epidemics, the hæmorrhagic diathesis complicates many cases, and they present the usual phenomena significant of profound alteration of the blood. Before the eruption makes its appearance, or subsequently, hæmorrhages take place in the skin from all the mucous surfaces, and into the parenchyma of organs. Profound adynamia sets in; the pulse is rapid and weak; the lungs are disabled by an extensive broncho-pneumonia; the abdomen is tympanitic, and profuse watery and offensive stools are discharged; the tongue is dry, the teeth covered with sordes; and low-muttering delirium ushers in death. A fatal result is not invariable, although usual in the hæmorrhagic form. The eruption may be absent in the mildest cases; it may pursue an irregular course, appear on the trunk before the face, remain on a very short time, or continue much longer than normal. Very high fever during the invasion stage, or great prostration, is significant of a severe case. The temperature furnishes the most certain guide to the actual state. Sometimes the eruption returns, the fever lights up, and all the phenomena of the disease are repeated. Various cutaneous eruptions may appear with the normal exanthem: as miliary vesicles, pustules, bullæ, and urticaria. Serious complications on the part of the eye must be noted—such as conjunctivitis, keratitis, iritis, etc. The larynx is the seat of ulcerations and erosions. Inflammation of the middle ear, succeeded by chronic otorrhœa, also takes place. But the most frequent and serious complications are capillary bronchitis, pneumonia, catarrhal pneumonia, etc. In some epidemics these complications are more numerous than in others, but the constitutional state and the hygienic surroundings are chiefly responsible. Capillary bronchitis and pneumonia occur during and after the stage of eruption. In strumous subjects catarrhal pneumonia may undergo the transformation into caseous, which is the explanation of the frequent occurrence of phthisis after measles. The constant association of enlarged follicles and intumescence of Peyer's patches in measles with the other morbid alterations characteristic of the disease, observed by the author in numerous autopsies, seems to justify his conviction that the former are really incident to the disease. An obstinate diarrhœa and dysentery (ileo-colitis) may occur at any point in the disease, but are especially troublesome from the period of retrocession of the eruption. Death is often due to this complication, or the convalescence is made very tedious. Simple uncomplicated cases of measles are free from danger. The indications that bode danger to life are an excessively high fever during the period of invasion; sparseness and dimness of the eruption while the general state is bad; confluence of the eruption and hæmorrhagic diathesis; anomalies in the development of the eruption, the other symptoms being unfavorable; capillary bronchitis, broncho-pneumonia, etc.; intestinal disorders, severe ileo-colitis, etc.; and cerebral complications.

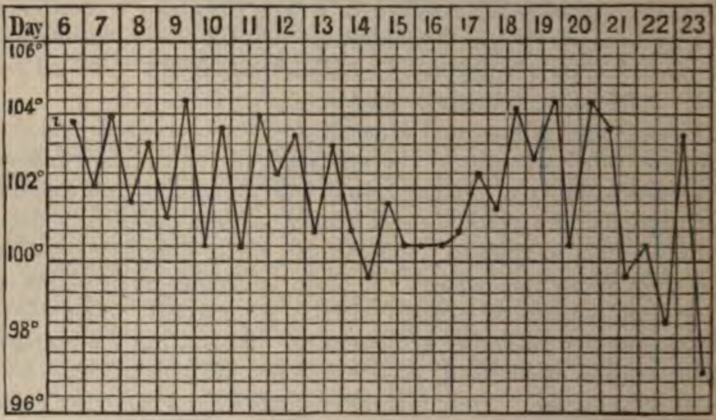

FIG. 54.—Range of Temperature in Measles complicated with Catarrhal Pneumonia.

Treatment.—Mild cases require confinement in-doors or to bed, on a regulated diet, and a little paregoric to quiet a troublesome cough. If the temperature is high during the initial stage, and the cough troublesome, a combination of aconite, ipecac, and opium is highly serviceable (tinct. aconiti rad., ʒ j, ext. ipecac., fl ʒ ij, tinc. opii deod., ʒ iij. M. Sig. Six drops every hour or two). If the aconite fail to reduce the temperature (the remission occurring during the invasion stage should not be overlooked), a tea- to a tablespoonful of infusion of digitalis may be given three or four times a day in addition. During the time of eruption, if the temperature is high, the skin should be rubbed every four hours with lard, or suet, or vaseline, or cacao-butter; and, if the fever is moderate, three times a day. If the bowels are confined, a simple saline laxative ought to be given. Free action of the kidneys can be maintained by cooling drinks. The temperature of the apartment should be about 70°, and, while it is well ventilated, all draughts must be excluded. The popular notion that measles requires a close room and blankets is a very pernicious one. The other extreme is equally dangerous. Such are the simple measures required in uncomplicated measles. When very high fever obtains through the prodromal stage, or subsequently, the antipyretic treatment most effective is the wet pack. The bed is protected by a rubber cloth, and over this is placed folded flannel of sufficient dimensions; a sheet wrung out in water, each time beginning at 95° and gradually cooled to 80°, is laid on the flannel; the patient is placed on the sheet and quickly wrapped up. This operation is repeated every half-hour until the heat is reduced. Besides the diminution of fever-heat, the wet pack develops the eruption, and exercises a most favorable influence on the course of capillary bronchitis and pneumonia, whence it is to be especially commended when the high temperature is the result of the pulmonary complication. Quite irrespective of the temperature, local wet packs are of very considerable importance in the treatment of measles. The vapor of water allays the nasal stuffing and the sneezing, and tepid-water compresses best relieve the irritation of the conjunctiva. Tonsillitis and laryngitis are much benefited by enveloping the neck in a tepid pack, and frequently renewing it. Packs and compresses are especially efficacious in the treatment of inflammatory affections of the chest and abdominal organs. If baths can not be utilized to reduce temperature, quinine comes next in point of efficiency, but it must be given in antipyretic doses. Antipyrin may be used instead of quinine, and digitalis can be combined with these antipyretics should a renal complication require it, and an irritable state of the stomach not prevent. In the hæmorrhagic form, quinine, the mineral acids, tincture of ferri chloridi, turpentine, etc., are especially indicated. The most important, as it is the most frequent complication, requiring careful therapeutical handling, is capillary bronchitis, with atelectasis, broncho-pneumonia, etc. The salts of ammonia, especially the carbonate and iodide, are of immense value in this state.

The plasticity and adhesiveness of the exudation are lessened by them, and thus the access of air to the alveoli is favored. They may be administered in an emulsion together, or the carbonate may be dissolved in solution of the acetate. The vapor of water is an important adjunct to the other means for lessening the obstruction of the tubes, and hence steam should be freely disengaged in the apartment. The volatile expectorants are very serviceable, in that they diffuse out of the blood through the lungs, and thus act locally on the affected surface. The most efficient of these are eucalyptol and turpentine, especially the latter, which is particularly indicated when the capillary circulation is feeble, the eruption pale, and the skin bluish. If the means resorted to fail to remove the obstruction in the capillary tubes, emetics become necessary. The subsulphate of mercury, alum, or sulphate of zinc, may be employed for this purpose—their repetition being determined by the results. Tartar emetic, which is often used, is greatly too depressing, and is dangerous. Apomorphine may also be given, but the remarkable effect which it now and then has on the heart is a serious objection to its employment. In the intestinal complication the author has had the best results from the conjoined administration of Fowler's arsenic (two drops) and opium (deodorized tincture, five to ten drops) every four hours, and from sulphate of copper and sulphate of morphine ($\frac{1}{20}$ to $\frac{1}{10}$ grain of the former, and $\frac{1}{16}$ to $\frac{1}{8}$ grain of the latter, for adults, three times a day). Very careful alimentation should be directed from the beginning, and should consist largely of milk, especially if there is a trace of albumin in the urine.

ROSEOLA—ROETHELN (GERMAN MEASLES).

Definition.—By the modern German authors the term *rubeola* is restricted to this disease, which is usually called *roseola* in this country. Following the course usually taken by American authorities, the term rubeola has been applied to true measles. Roseola is a self-limited eruptive disease, pursuing a course similar to measles.

Causes.—This is a disease of early life, appearing equally in the two sexes, and propagated by infection. It does occur in adults, but less frequently. One attack procures an exemption against future attacks, but this is not an absolute rule. That a peculiar materies morbi, virus, or germ exists is probable, but thus far it has not been isolated.

Pathogeny and Symptoms.—The eruption consists of rose-colored spots, the size of a pin-head up to three or four pin-heads, well defined and somewhat elevated, so that, when a number are placed near each other, the skin is distinctly rough. An hyperæmia of the papilla takes place, and of the adjacent cells of the derma above, and the redness in spots and the elevation of the hyperæmic patch give the impression of roughness. The spots have a round or somewhat oval shape, disappear on pressure, to return immediately when the pressure is withdrawn. The spots vary a good deal in size, and are rarely confluent or coherent. On the face, where they are most abundant, they do not flow together. They are nearly as abundant on the neck, chest, and abdomen. The eruption is quite abundant on the scalp, and extends freely over the extremities. The maximum development of the spots is about half a day, but the whole duration of their existence is from two to four days. A very slight discoloration remains for a day or two at the site of the spots, and very little, if any, desquamation takes place. From the period of exposure until the onset of the disease there are from ten to fourteen days. No symptoms occur until the eruption appears; in other words, there is no prodromal stage, or invasion, or initial stage. There is no fever in a majority of the cases. The eruption appears first on the face and spreads thence regularly over the scalp, body, and extremities, in about the same order as measles. A light grade of catarrh comes on with or immediately succeeds to the eruption, and there are redness, stuffing of the nose, sneezing, conjunctivitis, photophobia, etc., but all of these symptoms are much less severe than the corresponding symptoms in measles. More or less diffused redness, with punctations of deeper color, is observed in the mucous membrane of the fauces, pharynx, and larynx. Disorders of the intestinal canal or of the kidneys do not occur. In general the symptoms are so slight that children object to any restraint or confinement. Even in the few cases characterized by fever the symptoms are by no means severe, and the complications which occur are usually unimportant. The prognosis is favorable, and the treatment need consist in nothing more than confinement in-doors and intelligent supervision.

SCARLATINA—SCARLET FEVER.

Definition.—*Scarlatina* is an acute, infectious disease, self-limited, characterized by a peculiar exanthem, an affection of the throat and albuminuria, and terminating in desquamation of the epidermis.

Causes.—Scarlatina, like the other members of the group, is propagated by a peculiar poison, which, by reason of the tenacity with which it adheres to articles of clothing, and other peculiarities, we have good grounds for holding is a solid. It is communicated by contact of the healthy with the infected, and by intermediation of various substances to which the poison adheres. It occurs both in the sporadic and epidemic form, but never arises spontaneously. The susceptibility to scarlatina is not by any means universal, and is less than to variola and measles. The time which elapses, from exposure until the objective signs of the disease are manifest, varies greatly, and is therefore very differently stated by authorities. The shortest period is that of a patient mentioned by Trousseau, in whom the disease appeared in a day after exposure. The other extreme is twelve to fourteen days. The most usual period is from four to seven days. The very slightest contact with the morbific principle suffices. It may be conveyed on or about the persons of the healthy to others at a distance. That it may be dissolved in articles of food or drink is rendered highly probable by the epidemics following in the wake of milk distribution, of which several very instructive examples have been reported from England. The poison is probably contained in the skin and its excretions and epithelium, and also in the breath and exhalations from the throat. The period of greatest activity of the poison is at the highest point in the disease; but it is present at any period, from the initial to the terminal symptoms. The susceptibility varies greatly, even in members of the same family, hence nothing is more common than for one member of a family to be attacked while all the rest escape. The susceptibility to it is increased by all causes lowering the vital forces; and hence those situated under unfavorable hygienic conditions are more apt to be attacked. Again, the susceptibility of the same individual may vary at different times. Within the first six months of infant life there is little liability to the disease; but the susceptibility attains its maximum from the second to the fifth year, and declines slowly to the tenth, and after this more rapidly; but it does occur in old age. The author had under his care a gentleman of sixty years of age, with scarlet fever, after caring for several of his children with the disease, and his was a typical example. Sex and race appear to have no influence. Negroes are said to be less susceptible than whites. The author believes that this is not true, the misconception having arisen from the difficulty of recognizing the disease in the negro. The disease but rarely occurs twice in the same individual. Those exposed anew, especially if brought into close relation, as in the case of mother and child, are apt to suffer from the angina, without experiencing any of the other symptoms. Cases of recurring scarlatina are by no means infrequent; the author has seen two, in which, from one to three weeks after the close of the first attack, the whole phenomena of the disease were repeated, even to the desquamation.

Pathological Anatomy.—The eruption may be distinct, and around each spot a border of normal skin; or it may be confluent, the whole surface of a vivid red, with punctations of a somewhat deeper tint. The eruption is due to an intense hyperæmia, which is limited to the area of the spots, but which is general when the spots coalesce. At its first appearance the eruption is less vivid than it becomes when fully developed. The spots appear first on the neck and upper part of the chest, then on the face, where they are also most perfectly developed. They are nearly circular, are not elevated above the general surface, and do not therefore impart a roughness to the surface. They are also nearly equal in size, and when discrete uniformly distributed, about as much of the integument being covered by the eruption as free from it. When confluent the whole surface is a vivid, brilliant red, marked, as may be seen on close inspection, by minute points of deeper color. The eruption having reached the maximum of intensity, remains stationary from a half-day to a day, and then slowly declines. When the eruption first appears on the face, the redness of the temples, forehead, and cheeks contrasts vividly with the pallor of the lips. The eruption may be partial, or occur in particular localities, leaving large portions of the integument uninvaded. Thus, it may appear on the face only, on the trunk only, or on the extremities, especially around the joints. The individual spots may be two or three times as large as the usual eruption. A miliary eruption of minute vesicles may appear on parts so situated as to sweat freely, and a very fine papular eruption on all parts, notably on the forehead. In some cases the cutaneous appearances are diversified by hæmorrhages, and the formation of petechiæ and vibices. Other forms of eruption may complicate the scarlatinal eruption, such as herpes, urticaria, pemphigus, and other vesicular and pustular affections. As the eruption disappears, boils may be observed, and more or less gangrenous sloughing may occur in low states of the system, merely from pressure. Desquamation of the epidermis may succeed immediately to the eruption in a few days, sometimes in a few weeks, after it has disappeared. The exfoliation of the epidermis occasionally, in severe cases, takes place several times, and it is usually general over the body, but the intensity of the desquamation is not a measure of the intensity of the exanthem. The desquamation may consist of fine furfuraceous scales, and of large masses of exfoliation. The thick and hard epidermis of the hands and feet peels off in large flakes, and a cast of the hand or foot, like a glove or stocking, is not uncommon. Not unfrequently the hair and nails, and warts on the fingers, drop off. The skin is left red and sensitive by the desquamation, but its natural state is soon restored. Not less significant than the eruption is the affection of the fauces and of the pharynx. The mucous membrane of the fauces is intensely hyperæmic, of a deep-red color, and marked by conical elevations—swollen follicles—which rarely in simple cases suppurate and discharge. In the severer cases, instead of a simple redness there is a more or less deep, livid redness, involving not only the fauces, but the whole mouth to the lips, the pharynx, and the nares. Besides the deep coloration, there are increased secretion and œdema of the mucous membrane, especially of the soft palate. The tonsils are also deeply inflamed, much swollen, and are liable to form enormous purulent accumulations. There is a still more formidable affection of the throat, in which, besides the changes mentioned above, there are œdema of the throat, deep-seated inflammation of the tonsils, inflammation of the sublingual, submaxillary, and parotid glands, and simultaneous œdema of the areolar tissue of the neck, the whole forming a great mass of induration bulging out from the parotid region, and forming a broad band of induration filling in the whole space from the chin to the neck. The difficulties of the case are much enhanced by œdematous swelling and inflammation of the retropharyngeal connective tissue and that of the larynx. At the same time the tonsils may suppurate and slough, or become gangrenous, and from the tonsils the suppurative and gangrenous process may extend in all directions, and extensive abscesses form in the neck, followed by immense sloughing and loss of tissue. A diphtheritic process may also ensue in the fauces; and so common is it that a close relationship is supposed by many to exist between them. The tongue has a peculiar and very characteristic appearance. It is coated uniformly, except at the tip and edges, with a heavy whitish or yellowish-white fur, increasing in depth toward the base. Through this coating the enlarged papillæ project. On or about the third day an entire exfoliation of the coating, and of the epithelium also, takes place, leaving the surface of the tongue raw and red, and roughened by the elevated follicles, presenting the appearance of a fully ripe strawberry—whence the term "strawberry-tongue of scarlet fever." Troublesome affections of the ear occur with those of the throat. Inflammation of the middle ear, perforation of the drum, and in severe cases caries, preceded by periostitis of the squamous and petrous portions and of the mastoid process, take place. Also, in severe cases, the tissues about the ear externally are swollen, and pus dissects down the neck between the muscular planes. Inflammation of the larynx and œdema of the glottis during general dropsy, bronchitis, and pneumonia, are the lesions of the pulmonary organs occurring during the course of the severer cases of scarlet fever. Pericarditis, endocarditis, simple and ulcerative, with or without joint implication, are complications in many severe cases. There are no constant and uniform lesions of the digestive tube, pancreas, or spleen. The kidneys present, next to the skin and throat, the most constant anatomical changes. The tubules of the kidneys, like the skin, cast off their epithelium, which for a time may block the passages, until at length washed away by the urine (desquamative nephritis, tubular nephritis, etc.). Besides this, changes take place in the parenchyma (parenchymatous nephritis), already sufficiently described, succeeding to the other form, and occurring in the second to the third week. General dropsy and the accidents due to uræmia are usual concomitants of the kidney-disease. Closely connected with the condition of the blood due to the kidney-disease, if not dependent on it, are the attacks of inflammation of the serous membranes and of the synovial cavities of the joints. Meningitis, pleuritis, and peritonitis are the forms of serous inflammation, and acute rheumatism of synovial. The joint affection may consist only of a little pain and stiffness, or it may be a severe attack of rheumatism in which all the principal joints are affected in turn, peri- and endocarditis also occurring.

Symptoms.—By the older authors,* scarlatina was divided into *scarlatina mitis vel simplex*, *scarlatina anginosa*, *and scarlatina maligna* —scarlatina without any affection of the throat; scarlatina with decided implication of the fauces and adjacent lymphatics; scarlatina of the severest type with extensive suppuration, possibly gangrene. As these distinctions are rather artificial, we purpose describing first the ordinary, well-defined form, and mention subsequently the variations. *The period of invasion* is sudden and violent. A strong chill is the initial symptom in adults; in children, a violent convulsion or a succession of them, or a severe attack of vomiting, with prostration. Headache of a very intense character, general muscular pains and high fever succeed to the chill. In a short time the temperature rises to 104°, 105°, or higher; the skin is hot and mordicant; the throat burns, and, on inspection, the palate, tonsils, and pharynx are red and somewhat swollen; the tongue is coated with a thick yellowish fur. The fever is nearly continuous in type, and there are no strong remissions or intermissions, as in measles. The eruption makes its appearance usually at the termination of the first exacerbation of the fever—at the end of the first or beginning of the second day. It appears on the neck and upper part of the chest, and then on the cheeks and forehead, pale, rose-red, rapidly becoming brighter, and at first contrasting strongly with the white lips. Very quickly, in scarcely a half-day, has the eruption spread well over the body. In the more severe cases the eruption is not completed until the third or even fourth day. When the eruption is completed promptly, it is punctiform, each spot distinct and surrounded by an area of normal skin; when slower to reach its maximum, the eruption becomes confluent and diffused, the whole surface being of an intense scarlet hue. The tongue is thickly coated, but the coating with the epithelium peels off about the fourth day, leaving a red, raw surface, dotted with swollen follicles—the strawberry-tongue. There is no longer any vomiting, but the appetite is wanting, and there may be constipation or diarrhœa. Severe headache is experienced in the more decided cases; there are apt to be delirium at night and some confusion or somnolence through the day. On examination of the urine then, it is found to be scanty, high-colored, smoky, and contains more or less blood and albumin. The eruption is barely completed before it begins to fade on those parts where it first appeared—certainly, it does not stand at its maximum longer than half a day to one day. The gradual disappearance of the eruption is effected in two, three, or four days, and accordingly the time occupied by the eruption varies from three to seven days in its entirety. As the eruption fades away, the process of shedding goes on—at first, and for a short period, of a fine, furfuraceous desquamation, the shedding of large scales being subsequently the rule in most cases. The decline of the eruption is coincident with a diminution of the fever, and moderation of the general symptoms. The fever declines by lysis—by a gradual lengthening of the remissions and shortening of the exacerbations. The pulse subsides with the fever, the delirium ceases, the skin becomes moist, sudamina form, there is less and less trouble with the throat, and swallowing becomes easier; membranous exudations are cast off, the tonsils return to their normal size or nearly so, the tongue becomes moist, and its epithelium is reproduced; the appetite returns; the urine passes more abundantly, and carries off wasted and fatty epithelium, the albumin disappearing; and thus, in about ten to twelve days from the beginning of the symptoms, convalescence is established. But few cases, however, go through so mild a course. The points on which the scarlatinal poison may exert peculiar force are various. The degree in which the cervical glands are involved differs greatly. In the mildest there is simply some slight tumefaction of the lymphatics in the neighborhood; in the severest, the whole space between the chin and chest is filled in, extensive suppuration occurs, sloughing takes place, or more or less gangrene. Between these extremes there are numerous gradations of severity. The throat affection may be severe, and the exanthem light, and *vice versa*. It is sometimes the case that, when the throat affection is subsiding and the cervical glands are shrinking to the normal, a new disturbance arises in the glands; they swell to a considerable size, fever comes on, and convalescence is postponed. In the author's experience this reëxcitement in the cervical glands is secondary to an exacerbation of the renal troubles. Great differences also exist in the amount of the kidney complication. The absence of objective evidences of kidney-disease in the urine does not necessarily imply a healthy state of the kidneys. There are, however, very few cases in which a trace of albumin is not visible. When the hyperæmia of the kidney occurs, the urine, besides being scanty and acid, has a smoky appearance, from the presence of altered blood-globules uniformly distributed through it. On cooling, the urine usually deposits a great quantity of urates, cast-off epithelium (Figs. 39, 40, 43), and casts containing much of the tubular epithelium. The epithelium and casts are found at, or about, the time desquamation of the skin has commenced. The amount of albumin, when it first appears, is small. As the fever declines, and desquamation goes on favorably, the amount of urine discharged increases very much; it assumes a watery appearance and its specific gravity is low;

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* Gregory's "Lectures on the Eruptive Fevers," American edition by Dr. H. D. Bulkley, p. 151.

the albumin disappears, and in a short time the urine becomes normal. Parenchymatous nephritis usually develops during the desquamation period, in the third week, and rarely in the second. Then the urine becomes turbid from the presence of urates, blood-corpuscles, granular matter, casts, etc., is rather scanty and high-colored, and throws down a great quantity of albumin. No absolute rule can be laid down as to the period when the most pronounced renal symptoms will make their appearance, but the time named above must be regarded as usual. The occurrence of renal changes is the signal for other disturbances. The lymphatic glands of the neck enlarge very much, the appetite goes, and there are nausea, vomiting, and constipation, and sometimes a severe diarrhœa. Violent headache, disorders of vision, hallucinations, illusions, muscular twitchings, and eclampsia are experienced. The convulsions may be very violent in children, and one succeed another, with days of unconsciousness. The eyelids are swollen, and the legs pit on pressure. The urine may become very scanty, almost suppressed. The temperature may run very high, and the pulse be slow, falling to 60, 56, 50, and even lower, or the opposite conditions may prevail—the temperature may be below normal, and the pulse small, rapid, and feeble. As the symptoms become less grave, the urine flows more abundantly, but casts and epithelium may be present for some days, and albumin for weeks after the disappearance of any apparent disease.

Course, Duration, and Termination.—In the mildest form of scarlatina there may be a simple hyperæmia of the fauces, some swelling of the submaxillary glands, a transient fever of two or three days' duration, and the whole terminating in three or four days. In other cases there may be a pronounced rash, but no throat affection, no implication of the kidneys, and a few days of a mild fever, desquamation being almost entirely furfuraceous. But these mild cases may be followed by albuminuria and general dropsy, acute rheumatism, and other complications. Sometimes the case seems of the mildest character at the onset, but develops into a state marked by all the characteristics of a profound toxæmia. Others begin in that way. At the very onset, headache, delirium, convulsions, coma, tetanic cramps, and rigidity of the extremities, uncontrollable vomiting, severe dyspnœa, and a rapid, very feeble pulse, indicate the severity of the blood-poisoning, and death occurs in collapse before the eruption appears. As in every epidemic many of the mild, insignificant cases occur, so an occasional example of all that is most virulent in the scarlatina-poison is manifested in these cases, fatal within twenty-four hours of their appearing. On *post-mortem* examination no lesions of importance are to be seen, because the changes are of a subtile kind, occurring chiefly in the blood. During the course of a perfectly normal case of the disease, symptoms of a very formidable character may come on, consisting in sudden and great prostration of the powers of life, the pulse becomes extremely weak, the eruption fades, the skin grows cold, and death usually occurs in a few hours. In many cases, after a satisfactory course to the period of desquamation, the troubles growing out of a renal complication begin. There are differences in different epidemics as to the liability to this complication. When it occurs a general œdema ensues, and dropsical accumulations form in the great cavities, especially in that of the peritoneum. The urine is scanty, dark from the presence of blood, has a high specific gravity, and is heavily loaded with albumin. There are present vomiting and purging, dyspnœa from accumulation in the cavities, headache, somnolence, fever which varies in type, but is usually characterized by considerable remissions, the pulse being very slow and irregular. These cases of scarlatinal dropsy are usually quite fatal, not so much directly from the kidney-lesion, but indirectly from the pulmonary and intestinal complications. In other groups of cases, the specific gravity of the urine falls very low, and the quantity is also very scanty, and may be suppressed even for several days. Very formidable symptoms of uræmic intoxication arise under these circumstances, including defects of vision (amblyopia, amaurosis, albuminuric retinitis*), coma, convulsions (partial of muscles of mouth and extremities, trismus, and general). During such attacks death may ensue from the cerebral complications, by sudden œdema of the lungs, by exhaustion, etc. Although the prognosis is grave under these circumstances, remarkable recoveries from such states are noted during every epidemic. When a tendency to recovery exists, the stupor diminishes, the convulsions cease, the stomach becomes quiet, and food is taken, and the urine becomes abundant. There is a great tendency to relapse, and the change for the worse is often due to the use of solid and indigestible food. Even in those cases proceeding to a favorable termination, the urine is found to contain albumin in small quantity, after apparent entire recovery. These cases usually last from one to two or three months before entire restoration is accomplished. Other cases are remarkable for the persistently high fever, the extent of the throat affection, the severe intestinal troubles, and the cerebral complications due not to uræmia, but to the blood-poisoning. In these cases, which are often fatal, the result may be due to the consequences of the high temperature—reaching 106°, 107°, 108°, and even 109° Fahr.—while the pulse is at 200° ; to the obstruction to respiration in the condition of the throat ; to septicæmia, cerebral hæmorrhage, hydrocephalus, convulsions, etc. ; or to the exhaustion caused by extensive suppuration, sloughing, and gangrene of the throat, etc. The duration of such cases will vary from a few days to a week, or sometimes longer. *Recurrent scarlatina* is a form of the disease in which, after the whole process is completed and convalescence established, there occurs an entire repetition of the first seizure, including the desquamation. The second is somewhat shorter and less violent than the first attack. Another irregular form—to conclude the somewhat numerous varieties—is the hæmorrhagic. This is one of the most formidable varieties of the disease. The eruption is imperfectly developed, dark in color ; the throat is much swollen, and diphtheritic exudations occur, followed often by gangrene ; hæmorrhages take place from the mucous surfaces, from the kidneys, into the substance of internal organs, from the uterus, etc. These cases are uniformly fatal, death ensuing within the first week. Any prognostications in regard to the course and termination of a case of scarlatina should be guarded, for no disease is more uncertain. The case may be regarded as manageable when the initial stage is not severe, the eruption appears at the proper time, and attains its maximum on the second or third day, the throat affection is not extensive, the temperature never goes above 104° Fahr., and the pulse does not exceed 140 ; the cerebral symptoms consist only of a transient delirium at the highest point of the disease ; the temperature regularly and uniformly declines as the desquamation proceeds normally, and no other symptoms arise. Certain complications may exist without life being put in jeopardy. There may be mild complications of the kidney, and slight affections of the joints. The character of the epidemic is an important factor in the prognosis of individual cases. The mortality in different epidemics varies much —from ten to forty per cent.—and is determined largely, apart from the type of the epidemic, by the hygienic surroundings, and especially by age, infants succumbing in larger proportion than children and young adults.

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* "Die Albuminurie," etc., von Dr. Hugo Magnus, Leipsic, 1873, *op. cit.*

Treatment.—As scarlatina is a self-limited disease, and as we possess no specific against it, our treatment must necessarily be symptomatic. In directing treatment against the symptoms as they arise, we may select with advantage those remedies having a power to destroy ferments. During every epidemic there are numerous mild cases, which require only regulation of the diet, confinement, and supervision ; for the mildest cases may be followed by serious complications. For the initial fever, tincture of aconite-root (half a drop to a drop every hour, according to age, in a teaspoonful of water), and, preferably, the tincture or infusion of digitalis (from one to ten drops every two hours of the tincture, or five minims to a drachm of the infusion every two hours), are the most useful remedies. If the stomach is exceedingly irritable, and these remedies are rejected, a combination of carbolic acid and tincture of iodine is highly serviceable (℞. Tinctura iodinii, ʒ ij, acid. carbolic. ʒ j. M. Sig. One half a drop to one or two drops every two to four hours in water). If constipation exist at the same time there is vomiting, the usual condition during the initial stage, the most efficient laxative is calomel—from one sixth to one grain rubbed up with sugar and dropped on the tongue. During the period of eruption, should the surface be pale, the circulation feeble, and the eruption tardy in development, belladonna is the appropriate remedy (from two to ten drops of the tincture every two hours), or, if this fail, turpentine. If the temperature is very high during the eruption stage, and there is delirium, the kidneys acting freely, the wet pack is the most efficient remedy. If this will not be permitted, or is impracticable, the skin should be freely and often sponged with cold water, and rubbed with fat—lard, suet, cacao-butter, etc. In all cases when the eruption is abundant—is out freely—the fat should be used, the whole body in turn anointed every four hours. The effect of this is to allay the unpleasant heat and burning and to reduce the temperature. If vomiting continues during this period, the remedies indicated for this condition of things during the stage of invasion are appropriate at this period. Should diarrhœa be present with vomiting, an excellent means of arresting both consists in the use of bismuth and carbolic acid (℞. Bismuthi subnitrat. ʒ j ad ʒ ij, acid. carbolic. grs. ij ad grs. viij, mucil. acaciæ, aquæ menth. pip., āā ℥ j. M. Sig. A teaspoonful every two to four hours). If the throat complication is at all severe, the best method of treating it is to apply wet compresses, cold or warm, to the neck, enveloping it with several folds. The throat should be frequently gargled, if the age permit it, with hot milk-and-water, or pieces of ice may be held and allowed to melt slowly, keeping them well back in the mouth. Caustic applications should be avoided under ordinary circumstances. If sloughing and gangrene are taking place, strong solutions of nitrate of silver, the mineral acids, solutions of carbolic acid, and of permanganate of potassa, chlorate of potassa, etc., may be used. If there is much fetor, dilute sulphurous acid, iodine, and carbolic acid together, in solution, are effective, and may be freely applied to the fauces, and to all suppurating and sloughing surfaces. If there be active delirium during the eruption stage, the most appropriate medicaments for the relief of this condition are bromide of potassium, chloral (if the heart's action is good), morphine, and quinine in combination, if there is anæmia of the brain. During desquamation, the fat inunctions should be continued. Inflammations of the eye and of the ear occurring at this time should receive attention. Kidney complications demand treatment which shall be adapted to the condition present. If the urine is scanty, bloody, and of high specific gravity, if there are pain in the back and strong pulse, leeches or cups should be applied to the lumbar region. Large draughts of water or of milk, milk and lime-water if the stomach is irritable, cream-of-tartar lemonade, infusion of digitalis, topical application to the lumbar region of digitalis, pilocarpine, etc., are the most appropriate remedies. For further particulars of the treatment of the kidney complication, the reader is referred to the sections treating of these diseases. For those cases exhibiting profound alterations of the blood, the remedies possessing anti-ferment powers, as carbolic acid, salicylic, benzoate of soda, thymol, etc., may be employed. The most useful of these, the author believes, is the combination of carbolic acid and iodine, already mentioned. Extraordinary results have been claimed for the carbonate of ammonia, and equally confident claims have been put forward for yeast. The character of epidemics varies so much that caution is necessary in accepting the conclusions of over-confident therapeutists.

DIAGNOSIS OF VARIOLA, VARICELLA, RUBEOLA, ROSEOLA, AND SCARLATINA.

To avoid repetition, and to make the differentiation as clear as possible, the question of the diagnosis of the above diseases has been postponed until they have been considered in the regular way. They may be compared in their period of invasion, stage of eruption, and stage of desquamation.

Stage of Invasion.—In small-pox the duration of this stage is three days, or until the third exacerbation of the fever; in measles, four days or longer; in scarlatina, one day or two. In measles there is a strongly marked remission at the end of the second or the beginning of the third day—in small-pox there is no such remission; in measles the temperature does not decline at the appearance of the eruption—in small-pox there is a marked remission or an entire cessation of fever when the eruption appears; in small-pox the stage of invasion is often diversified by rashes and there is no coryza—in measles there is coryza but there are no initial rashes. The invasion stage of scarlatina differs from small-pox in duration, in the absence of any initial rashes, in the higher temperature, in the coincident angina, and swelling of the lymphatics.

Stage of Eruption.—The eruption of variola is first red spots, then papules, then vesicles, and finally pustules, and they appear first on the face, forehead, and head; that of measles is reddish, lenticular spots, slightly elevated above the skin, and imparting a sense of roughness to the surface; that of varicella, vesicles; that of roseola, rose-red spots like measles, but not so prominent; that of scarlatina, bright-red spots and diffused redness, with punctations of deeper red. The eruption of small-pox on its appearance has an indurated feel, as of a solid body—a bird-shot—in the skin; that of measles imparts a sense of roughness wholly on the surface; that of varicella has to the touch the sensation of a vesicle elevated above the surface; and that of scarlatina has no roughness, but is a vivid scarlet-red spot, which disappears on pressure, to return as soon as the pressure is removed. The eruption of small-pox requires many days to develop, and its maturation is accompanied by distinct fever; that of measles, roseola, varicella, and scarlatina reaches its maximum in a day or two. The eruption of measles is accompanied by coryza, watering of the eyes, a coarse, bronchial cough—that of scarlatina by sore-throat and swelling of the submaxillary and sublingual and cervical glands; both desquamate—the former in fine, furfuraceous scales, often not perceptible—the latter in large flakes and very distinctly. The pustule of small-pox forms a distinct crust and leaves a scar; that of varicella dries up and drops off without a mark. The eruption of measles differs from roseola in that the former is darker in color, is accompanied by fever, coryza, etc., not present in the latter.

Stage of Desquamation.—Desquamation occurs in both measles and scarlatina, but differs greatly in thoroughness, as is above stated. The complications of this period are, in scarlatina, affections of the kidneys, dropsy, uræmia, etc.; of measles, catarrhal pneumonia, capillary bronchitis, and ileo-colitis. Desquamation does not occur in small-pox until the pustules have matured and crusts formed.

ERYSIPELAS.

Definition.—*Erysipelas* is a self-limited, febrile affection, characterized by a local inflammation of the skin, terminating in desquamation, and accompanied by constitutional symptoms and the usual phenomena of blood-poisoning.

Causes.—The most influential factor in its propagation is contagion. It prevails in hospitals, and epidemics follow in the paths of armies. A peculiar poison, it is assumed, enters a wounded surface, and, after a certain period of incubation, the phenomena of the disease follow (Trousseau). Nevertheless, the disease has been divided into two classes—*idiopathic* and *traumatic*—the former arising spontaneously, the latter in connection with a wound. That this distinction must still be maintained is probable, because there are many cases of erysipelas for which there is no traumatic cause, and which must be, therefore, idiopathic. It is asserted that women are more susceptible to the poison than men; but later researches have shown the incorrectness of this statement. It is a disease of all ages, but is rather more usual from the twentieth to the forty-fifth year of life. It occurs at all seasons, but is more prevalent during the variable weather of winter and spring. The author has witnessed two epidemics of erysipelas and puerperal fever, occurring together, and acting apparently in substitution.

Pathological Anatomy.—The whole thickness of the skin is involved, and the inflammation extends through to the subcutaneous connective tissue. The derma is bare by exfoliation of the epidermis and uppermost cells and the papilla; and the connective tissue, with the sweat and sebaceous glands, is œdematous and infiltrated with white blood-corpuscles in great numbers. By the accumulation of cells an abscess forms at the summits of the papillæ. As soon as the redness in the skin subsides, the cells thickly distributed through the subcutaneous tissue undergo a granular disintegration; a portion of the detritus thus produced enters the lymph-vessels, and the rest is absorbed, leaving the skin normal. Various changes have been reported as occurring in internal organs; but little definite information exists in regard to them, except granular degeneration of the heart and vessels, the liver, kidneys, and spleen, which appears to be definitely established. The blood seems to be much changed, but the reports are not uniform as to the character of the alterations. Bastian has ascertained the existence of capillary embolisms of the cerebral vessels, in some cases of death, from erysipelas of the face.

Symptoms.—Like the other eruptive fevers, erysipelas sets in by a stage of invasion. The initial symptom is a chill, although not usually a violent chill. Headache, often of an intense character, comes on with the fever; and there are nausea, bilious vomiting, and entire loss of appetite. Before the eruption appears, and thus directing the diagnosis, some of the cervical lymphatics, or the submaxillary gland, swells—the former when the erysipelas appears on the head, and the latter when it attacks the face. That this sign shall be available, the initial stage must be longer than a half-day. A sense of heat and tension is felt in the skin which is about to become inflamed. A patch of redness appears, and at several points, which coalesce and thence spread widely. The red color disappears on pressure, to be quickly restored; but, when the red disappears, a yellowish rather than white hue is seen. The skin, inflamed, is also œdematous, and it presents a tense, shiny appearance. The redness may commence at any point on the face or scalp, but it usually takes its origin from some accidental abrasion or from a pathological lesion, as a patch of eczema, or impetigo, etc.; and, when not initiated by such cause, it is apt to begin at or near one of the cavities opening externally—at the mouth, nose, or meatus auditorius. It was the opinion of the late Dr. Todd that many cases of erysipelas begin in the fauces and spread thence to the lips and elsewhere. The appearance of the eruption is accompanied by a sensation of heat, burning, and tension, and sometimes there is acute pain in the affected part. Where the parts are lax, and the exudation has room, there is less pain, and the swelling, therefore, is inversely as the pain. When there is great distention, and also abundant and rapid exudation, the epidermis is raised into blisters of varying size, according to the state of the skin. These blisters contain a transparent serum; sometimes they are reddish from the presence of blood, or yellowish from the number of pus-corpuscles, and they contain great numbers of bacteria. Where the cellular tissue permits, the swelling may be enormous, and the head and face so transformed that not a single feature is recognizable; the eyes can not be opened, the nose is closed, and the lips so stiff and swollen as scarcely to permit of feeding. The inflammation reaches its highest point on the second or third day, when the retrograde process begins, and on the fourth, fifth, or sixth day the redness is fading and the color is becoming yellow, and less and less swelling is noted. The blebs dry into yellow scabs or crusts. Suppuration may take place at various points after the termination of the inflammation in the skin, but the pus is usually absorbed without difficulty. Desquamation of the epidermis takes place over the whole area occupied by the inflammation, and the hair drops out, to be, however, quickly reproduced. During the maximum of the inflammation the scalp is very tender, and much pain is experienced wherever the head rests. The great peculiarity of erysipelas is its migratory character, spreading widely from the point where it first appeared to distant parts of the body. The margin of the redness is not sharply defined, but the swelling forms an abrupt ridge. The diffusion of the inflammation is not a mere chance, but pursues its course along the lines of least resistance, as determined by the arrangement of the fibrous-tissue bundles. The opinion of Todd, that erysipelas may start from an inflammation of the fauces, is supported by Trousseau and other authorities, and the erysipelas may extend downward into the glottis. The mucous membrane may also be attacked secondarily by extension of the inflammation from the skin. A heavily coated tongue, whitish or yellowish-white, becoming blackish, and ultimately peeling off in large flakes, is the condition of this organ. There are usually much nausea, protracted vomiting, entire loss of appetite, and excessive thirst. The intestinal evacuations may be normal, or diarrhœa may be present, or black, foul-smelling, unhealthy discharges may occur. Ulcerations of the duodenum, and consequently intestinal hæmorrhage, are by no means uncommon. The urine may contain albumen and casts, and indeed a small quantity of albumen seems an invariable result; hence uræmia, with all its possibilities, may enter into the symptomatology of erysipelas. There are few cases of severe erysipelas without some transient delirium. Often there is active delirium during the highest point in the case. There are three chief sources of the delirium: cerebral anæmia, a reflex result of the cutaneous inflammation; alcoholic excess; thrombosis of the capillaries, or sinuses. The two first named may or may not be important; the last is probably always fatal. Fortunately, it is rare. It was Bastian, we believe, who first pointed out the capillary thromboses resulting from facial erysipelas. The explanation is afforded by the intimate anatomical connection of the facial vein with the pterygoid plexus and cavernous sinus. Delirium is also a result of continued high temperature, but more especially a result of a combination of high fever with cerebral anæmia, the patient one who had been addicted to alcoholic excess. At the onset of the inflammation the fever may reach 104° or 105° Fahr. The type of the fever is remittent, and a rapid defervescence ensues usually about the fourth, fifth, sixth, or seventh day; but this defervescence is determined by the cessation of the inflammation in the skin. If the eruption continues to spread, there will be fluctuations in the temperature corresponding to the varying condition of the skin. The pulse varies accordingly and ranges from 100 to 140.

Course, Duration, and Termination.—Erysipelas corresponds to the other eruptive diseases, in its tendency to spontaneous cure at a certain period, but this is less certain, owing to its erratic course over the skin. The usual duration is from one to three weeks, but it may continue much longer when it tends to spread over a large part of the integument. When it ceases, in that which may be regarded as the typical mode, on the fourth, fifth, or sixth day, by a rapid defervescence of the temperature, there often occurs some critical evacuation—a profuse sweat, free intestinal movements of a very offensive character, or a large urinary evacuation; but these critical phenomena are not always present. Primary or idiopathic erysipelas, notwithstanding the horrible aspect presented by the patient and the occurrence of considerable delirium, usually terminates in recovery. The convalesence is rather tedious because of the low condition to which the patient is reduced, even in favorable cases. There are dangers, fortunately rather rare, which attend the primary form of the disease—the occurrence of thromboses, capillary or of the sinuses; the formation of ulcers in the duodenum; the extension of the inflammation to the fauces; and the depression of the powers of life, which may coincide with the sudden defervescence of the temperature. The traumatic form is more serious, because the erysipelatous inflammation is added to the complications of the injury. Furthermore the local hygienic conditions surrounding the wounded are favorable to the development of serious complications. Erysipelas coming on during convalescence from such serious diseases as typhoid, pneumonia, diseases of the heart, diabetes, etc., is always a very dangerous malady. On the other hand, important complications may arise during the course of an ordinary erysipelas. Thus a pneumonia, pleuritis, peritonitis, or meningitis, may arise by extension of the disease. Although the connection between the external malady and the disease within can not always be traced, it probably exists. Finally, an attack of erysipelas may terminate in pyæmia.

Diagnosis.—Erysipelas may be confounded with erythema, urticaria, and with phlegmonous erysipelas. Erythema is a superficial redness without inflammation—without heat and swelling—is without fever, and does not desquamate. Urticaria occurs in the form of wheals that itch a good deal and disappear in a few hours. Phlegmonous erysipelas, so called, is a deep-seated inflammation, with suppuration, spreading along the connective tissue and by the intramuscular planes from a wound or injury, and does not take the course along the integument as erysipelas. So characteristic are the appearance and behavior of erysipelas that it would seem impossible to mistake it for any other disease. The diagnosis by anticipation should not be overlooked —the occurrence of enlarged lymphatics in the neck in the case of erysipelas of the scalp, and of enlarged submaxiliary glands in the case of erysipelas of the face.

Treatment.—The perturbating treatment formerly used is now no longer employed. The mildest cases require only a laxative, a suitable diet, and the local application of some vaseline to abate the heat and burning. In the more severe cases there can be no doubt of the value of quinine, especially if combined with belladonna. To avoid the complications which may arise in even simple cases, the author gives the tincture of belladonna, or preferably a solution of atropine (atropinæ sulph. gr. j, aquæ ℥ j. M. Sig. One drop every four hours in some water). As the effect of the atropine accumulates, the interval between the doses is enlarged. In the more severe cases quinine should always enter into the treatment, and in full medicinal not antipyretic doses (℞ Quininæ sulph. ℈ij, ext. belladonnæ gr. iij. M. Ft. x pil. Sig. One every four hours). The delirium of anæmia, the usual form, especially in those addicted to alcoholic excess, is best relieved by alcoholic stimulants, and morphine and belladonna, if the latter does not enter into some other combination. The systematic use of milk and beef-essence is necessary in all severe cases, especially under the conditions named above. Tincture of chloride of iron, in half-drachm doses every four hours, is much commended by the English physicians, and with good reasons. In traumatic erysipelas Mr. Higginbotham's mode of applying a solution of silver nitrate in nitric ether is most serviceable. The surface must be carefully washed and dried. Then the following solution is brushed over the inflamed area, and for a short distance beyond on the healthy skin. On drying, should any part of the skin appear untouched, the solution is reapplied to these parts. The usual strength is about as follows: ℞ Agenti nitrat. ℈j, spts. ætheris nitrosi ʒ ij. M. Sig. Apply with a brush. An aqueous solution of two drachms to the ounce may be employed instead. The topical applications recommended are almost innumerable—a fact which indicates the uncertainty of value of any article. As a rule, irritating applications do more harm than good. To this dictum must be excepted the application of nitrate of silver, in the traumatic form of the disease. The author has seen murcurial ointment, diluted ten times with lard, very successful. Probably still better is the following: Vaseline ℥ j, acid. carbolic. ʒ ss., or less, which should be brushed over the inflamed area three or four times a day. Above all remedies and applications is the use of a nutritious diet. From the very beginning systematic feeding should be carried on. When the patient can retain nothing else, lime-water and milk may be retained. But, when the stomach becomes quiet, milk, eggs, animal broths, etc., should be given at regular intervals, and, when necessary, stimulants. Trousseau (*op. cit.*) used no remedies except a laxative, but he pushed the administration of food, and of the great number of cases treated by him only three died.

FEVERS.

TYPHOID FEVER.

Definition.—*Typhoid fever* is an acute febrile affection, self-limited, feebly if at all contagious, and characterized by a peculiar eruption on the abdomen, by a form of diarrhœa, by stupor and low delirium, by thickening and ulceration of Peyer's patches, by infiltration and softening of the associated mesenteric glands, and by swollen spleen. Various names have been applied to this disease. In Germany * and France,† and on the Continent generally, it is now called "abdominal typhus"; in England and this country it is usually designated *typhoid* —rarely *enteric fever,* the term which was originally proposed by the late Professor George B. Wood. Notwithstanding the term *typhoid* is excessively faulty, it is so universally used in this country that the author has adopted it.

Causes.—Typhoid owes its origin to a peculiar poison, whose source and nature have thus far eluded investigation, but is associated with the decomposition of animal matter under certain conditions. It is never produced by mere decomposition of animal matter, fæces, or the contents of sewers; it is essential to the formation of the poison that the typhoid germ be present, and this germ finds in these decomposing animal matters a suitable soil for its growth and development. It does not originate *de novo,* but there must be present some typhoid matter furnishing the material for a new growth. There are sound reasons for concluding that the poison is contained in the excrements, but it seems necessary for some change to go on in them to develop the activity of the poison, for when in the fresh state they manifest no activity. Admitted to the cesspool, or to the sewer, or the on the ground, the germ becomes active and multiplies, so that the excretions of a single patient may develop sufficiently to poison a large community. The germ of typhoid is extremely viable, and preserves its activity for a long time, so that, should typhoid occur in a given locality and then disappear for a long time, another epidemic may develop without the introduction of a new case. How long the poison may remain in the body before the advent of symptoms may not be very definitely established. The average duration of the incubation period may be stated at three weeks, although it may be as short as one week, or as long as four. The vehicles by which the disease-germs reach the body are air, water, articles of food, etc. In the gaseous exhalations from the sewers and privies, the *materies morbi* is carried up and is inhaled; dissolved in drinking-water or in milk, it is conveyed into human stomachs; and it may be deposited on other articles of food to be similarly disposed of. That the *materies morbi* does not infect a larger number is probably due to the insusceptibility of many persons receiving it. Susceptibility to the poison is developed by various influences. The seasons have the power to modify the prevalence of the disease. In this country the fall and winter are seasons of the greatest prevalence of typhoid. Loomis * says it is most prevalent in autumn, whence it is known as "autumnal fever." The condition of the water-supply, as to its elevation, Buhl has shown for Munich, is an important element, and that typhoid decreases as the water rises, and increases as the water falls. Age affects the predisposition to typhoid, and the tendency to it is greatest from fifteen to thirty years; it is almost absent in children under one year and in the aged. Men are rather more susceptible than women, and the disease selects by preference the most vigorous and able-bodied, and passes by those suffering from chronic diseases. One attack furnishes exemption against those in the future, but this rule is frequently violated. Recurring typhoid, however, like recurring scarlatina, is not uncommon.

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* "Handbuch der Pathologie und Therapie des Fiebers," von Dr. C. Liebermeister, Leipsic, 1875, p. 690.

† "Traité de Pathologie Interne," par S. Jaccoud, Paris, 1871.

Pathological Anatomy.—The lesions of typhoid fever are eminently distinctive. The extent of the changes, although, as a rule, indicative of the violence of the attack, is not always so; for with comparatively few lesions there may be formidable symptoms, and *vice versa.* As it is probable that the poison enters the intestinal canal, and there begins its ravages, it is most appropriate to begin the sketch of the morbid anatomy with the intestinal lesions. The title *dothiênentérie,* first given to this disease by Bretonneau, and adopted by Trousseau,† was intended to emphasize the importance and particularity of the intestinal lesions. Conforming to the method of Trousseau and of Liebermeister, who pursued a chronological arrangement, we may divide the appearances into periods of weeks. In the first week there are more or less hyperæmia and swelling of the mucous membrane in the ileum at its lower part, and especially around the patches of Peyer. Coincidently, a few of Peyer's glands and some solitary follicles are swollen by infiltration, especially those glands near to the ileo-cæcal valve, and by the end of the first week the infiltration has become general. The congestion is not limited to the mucous membrane, but often extends to the peritoneal surface, which is intensely hyperæmic (Lyons*), to the mesentery, and to the spleen. In the second week occurs the infiltration of the glands of Peyer, and the hyperæmia lessens. Stimulated, we may suppose, by the typhoid-poison, the cellular elements of the glands, agminated and solitary, undergo a rapid proliferation, by multiplication of their nuclei and by division. This increase of their contents causes them to swell in all directions, so that they rise above the general surface of the intestine, and appear dark or reddish. The solitary follicles vary in size from half a line to a line in diameter, to the volume of a small pea, and may even reach the dimensions of a bean, while the patches, oval in shape, are elevated above the surface, from one sixteenth to one quarter of an inch. The new cells are not confined to the glands entirely, but wander forth, infiltrating the neighboring mucous membrane, and, passing through the muscular, penetrate to the subserous layer. At and near to the ileo-cæcal valve, a number of the patches cohere and unite, forming oblong masses, and even surrounding the valve with a ring. The patches also coalesce at the extremity of their long axis, parallel to the long axis of the intestine, and thus attain extraordinary length. The number infiltrated is not always the same; they may all be involved to a greater or less extent; there may be but three or four. The same differences exist in respect to the number of solitary follicles infiltrated. The rapid and large production of new cells imparts to the glands and follicles a soft, spongy character, and soon leads to a necrotic softening and sloughing. It is, however, in the more pronounced cases that the patches become necrotic. They have usually a greenish color, from the presence of bile-pigment, or are stained a brownish color by the intestinal juices. The sloughs are cast off during the second week, leaving an ugly excavation which reaches to the muscular coat, and often to the serous. These ulcers have the shape and size of the involved patches, and are elliptical in form, their long diameter parallel to that of the intestine, and their margins are thick and sharply defined. Enormous ulcers may form in the neighborhood and around the valve, by the union of many; indeed, this part may be a mass of ulcerations, with small bits of mucous membrane between them. The process of ulceration and necrotic sloughing may be postponed to a much later period. Several months, indeed, may be occupied in the process of typhoid infiltration, without ulceration taking place (Lyons). But such examples are clearly exceptional. When extrusion has taken place, the process of healing goes on in favorable cases. The floor of the ulcer is soon covered with granulations, and, a gradual contraction taking place, the ulcer is ultimately closed, a cicatrix marking the site. A restoration to the normal is accomplished in many patches and follicles without ulceration. The new cells disintegrate and disappear, the hyperæmia subsides, and the original state is resumed. It is probable that this is the course of the lesions in the mildest cases. The two processes are usually mixed. Amid more or less extensive sloughing and destruction of substance, there will be seen patches and follicles that do not ulcerate, and whose new elements degenerate and are absorbed. The part of the intestine affected has an influence on the result—the sloughing and ulceration taking place below, and the retrogression by degeneration and absorption occurring above. The process of sloughing and repair may go on together, and at a very advanced period, so that perforation may result when healing is far advanced.

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* "Lectures on Fevers," by Alfred L. Loomis, A. M., M. D., New York, William Wood & Co., 1877, p. 403.

† "Clinique Médicale," tome premier, Paris, 1865, p. 212, *et seq.*

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* "Treatise on Fever," Philadelphia, 1861, p. 362.

It has already been mentioned that the initial hyperæmia involved the mesentery as well as the intestines. Other changes occur in the mesentery, following in the wake of those going on in the intestine. The glands swell, are congested, reddish, and succulent. They enlarge very considerably by an accumulation of their contents, and attain the size of a bean, an almond, or a pigeon's-egg. They presently soften, and many become diffluent and are barely retained within the capsule. When retrogression takes place the soft material is absorbed, the congestion disappears, and the glands shrink to their normal size. Sometimes a purulent collection remains behind, and a slow, cheesy transformation is effected. It not unfrequently happens that other lymphatic glands are infiltrated to some extent, such as the retroperitoneal and bronchial glands, etc., but in the mesentery the glands usually attacked are those immediately related to the diseased part of the intestine, although in severe cases all may be swollen and infiltrated. The spleen is affected in a similar manner. When the hyperæmia begins in the intestines the spleen enlarges, and by the end of the first week the enlargement is sufficient to be recognized through the abdominal wall, and at the maximum the organ is two or three times larger than normal. The change consists in a multiplication of the cellular elements, which at first increases the firmness of the organ, but ultimately it becomes exceedingly soft, so that it almost falls to pieces by its weight. The retrogression occurring in the spleen consists of a degeneration and disappearance of the new elements; the capsule contracts, the trabeculæ become more firm, and the pulp more compact. The lesions thus far considered are peculiar to typhoid. We have now to discuss changes due to a persistent elevation of the temperature, and known under the designation of parenchymatous degeneration of organs—the liver, kidneys, muscular tissue of the heart, the nervous system, and muscular system of animal life in general. Parenchymatous degeneration is a granular and fatty change affecting the proper gland elements. In the liver the cells become clouded with fat-granules and the nuclei disappear, and when the change is most advanced they break down into granular fragments. The effect of this process is to diminish the firmness and consistency of the organ, to change its color to a grayish or yellowish-red, and to materially diminish the blood in the small vessels. The degree of the change varies chiefly in accordance with the range of temperature ; it may be very slight or very considerable, and the right lobe is usually further advanced in the change than the left. In the kidneys the epithelium of the tubes, first of the cortex, then of the pyramids, becomes granular, cloudy, and the contour indistinct, the nuclei disappearing, and at last breaks down into granular fragments. The effect of these lesions is to diminish the firmness of the organs, and to change their color. In the kidney, as in the liver, the amount of the change varies, and is determined by the range of temperature. Albuminuria results when the alteration is at all extensive. Very important are the changes occurring in the cardiac muscle. The granules appear in large numbers, arranged in parallel rows, filling the fibers, and ultimately causing a disappearance of the striæ. The result of this change is very injurious. The tissue of the heart is soft, flabby, and easily torn, and the organ in advanced cases can not maintain its shape when laid on a table, but flattens out like so much mush. In the muscles the degeneration takes the two forms of granular and waxy. In the brain the changes due to parenchymatous degeneration have not been, as yet, adequately studied, but the naked-eye alterations are very definite, the chief change consisting in anæmia, œdema of the brain, the subarachnoid spaces, the perivascular lymph-spaces, and the ventricles containing a good deal of fluid. Some parts of the brain are less firm than normal, and more or less atrophy occurs, the convolutions flattening and the ventricles enlarging, etc. Rarely the lesions of an acute meningitis are superadded to those of typhoid. In the respiratory organs there are various lesions, which, if not essential to typhoid, are at least usually associated with those that are peculiar. Not unfrequently the larynx is attacked with ulceration ; but the most characteristic change is that of catarrh of the bronchial mucous membrane, which is swollen, deeply injected, and coated with viscid mucus. The access of air being cut off from some of the vesicles, they collapse, or pass into the state of atelectasis. The dependent portions of the lungs are in the condition of hypostasis, with or without œdema, and in rare cases lobar or lobular pneumonia.

Symptoms.—As a rule, a prodromic period ushers in a case of typhoid. For a week or ten days, or even longer, a lack of the usual vigor and a disposition to tire easily are perceived. Headache, epistaxis, tinnitus aurium, a poor appetite, and a slight diarrhœa, are also noted. The mind is dull, and mental application is very fatiguing; sleep is disturbed by dreams and is unrefreshing. Presently some chilliness is felt at different times and for several days, and the fever begins; the strength is exhausted, and the patient betakes himself to bed. In other cases, the prodromic period is characterized by the development of an acute catarrh of the stomach; there are disgust for food, nausea, and a heavily coated tongue; temporary relief is afforded by spontaneous or contrived vomiting, but the symptoms are soon resumed, the nausea continues, some diarrhœa occurs, great weakness is felt, headache, hebetude of mind, and disturbed sleep are experienced, and gradually the fever lights up. In still other cases—and they are relatively very numerous in the malarial regions of this country—an attack, apparently of intermittent fever, precedes the fever proper; there may be several distinct paroxysms, but the fever soon assumes the remittent type, and the phenomena of typhoid gradually develop. A few cases begin without any prodromes. A person, apparently in full health, is unexpectedly seized with some chilliness, followed by fever, languor, headache, etc. On the next day there is more chilliness, the fever is more pronounced, the mind is already becoming dull, and the other symptoms of typhoid come on immediately. The disease is held to originate with the first chilliness or the first elevation of temperature, and from these data is computed the duration of the different periods. As the appearance of fever marks the onset of the disease, so its decline and disappearance establish convalescence.

First Week.—The symptoms of the prodromic period are more pronounced: there are violent headache, a sense of confusion and mental weakness; singing and drumming in the ears; some bleeding at the nose, often but a few drops escaping; the eyes are intolerant of light, the ears of sound; the patient may still get on and off the bed, but, when he attempts to stand erect, his limbs tremble, and he is seized with vertigo. The appetite is gone and the suggestion of food is repugnant; there is a bad taste in the mouth, and the thirst is excessive. The tongue is at first large, pale, indented at the margins with the teeth, but it becomes dry and smaller by the fifth day; the coating peels off with the epithelium in patches, leaving a very red, dry, and glazed surface, and it is also somewhat tremulous. Some diarrhœa may have existed during the prodromic period, and there is often a tendency to constipation during the first week, but, when this is the case, it is found that a light purgative acts with unwonted violence. More or less diarrhœa exists during the first week. At first the stools consist of thin, brownish fæces, having a rather strong odor, but they increase daily in number and change in character toward the end of the second week, when they assume the yellow ochre color, the well-known "pea-soup" appearance. When they are permitted to stand, they separate into two distinct strata: the upper one liquid, holding salts, extractives containing bile, epithelium, ammoniaco-magnesian phosphate, and fat, finely emulsionized; the lower one, more consistent, containing analogous ingredients to those in the upper layer, but, in addition, a quantity of soft, yellow concretions made up of fat, albumin, pigments, and phosphates (Jaccoud). Great interest attaches to the microscopic examination of the stools, since it is generally conceded that the typhoid matter exists in the stools, but the results thus far attained can not be regarded as conclusive, although various microbes have been described by Klebs* and others as the specific infective element, or typhoid germ, but until their infective power is demonstrated their relation to typhoid fever must continue conjectural. Gurgling in the right ileo-cæcal region has been classed among the symptoms of this period, but, as it is present in diarrhœal affections, there is no great value to be attached to it alone. Tenderness, as well as gurgling, makes a more significant impression, especially if, as there ought to be, at the end of the first week, some fullness, even distention of the abdomen. At this time distinct increase in the area of splenic dullness can be made out, and the enlarged spleen may be often felt. Enlargement of the tonsils, follicles of the pharynx, and of the large follicles at the base of the tongue takes place coincidently with the development of the intestinal glandular appendages. Catarrh of the bronchial tubes, shown by some dry and moist *râles* over the dependent portions of the lungs especially, comes on at this time, but its intensity varies in different epidemics and in different individuals. At the end of the first, or at the beginning of the second week, appear the very characteristic disorders of the nervous system. The restlessness, the complaints about the aching in the back and limbs cease, and instead there is a condition of apathy and indifference. The patient becomes somnolent, but is easily aroused, and does not sleep well at night. Some of his indifference and stupidity of expression is due to dullness of hearing, and hence he must be spoken to somewhat loudly. When roused he responds correctly, and expresses himself as feeling very well. From the seventh to the tenth day some disturbance of mind is noted; it may be toward evening, or at night only, or when roused, and ordinarily it is nothing more than a tranquil muttering, or, as it is commonly expressed, "low-muttering delirium." Sometimes the delirium takes a more active character: it is wild, furious and ungovernable; the patient gets out of bed, resists the attempts to feed him, spits his drink or medicine into the face of his nurse; will not keep any covering on him, talks incessantly, and not only gets out of bed, but will jump out of the window. This condition of wild delirium coincides with greatly elevated temperature, rapid pulse, and the other evidences of extreme illness. Fortunately, these cases are rare. Usually the delirium is a low monotone, mumbling incoherently, and is accompanied by picking at imaginary objects on the bedclothes, and subsultus tendinum. The trembling of the muscles is seen not only in the subsultus of the tendons of the forearms, but in the protrusion of the tongue. If not too far gone in stupor, the patient may yet protrude his tongue when urged to do so, but he does it slowly, hesitatingly, with much trembling, and he forgets to return it again, keeping it protruded until forced to return it. The urine is acid in reaction, is rich in urea, urates and extractives, and poor in chlorides. The urine frequently contains the urinary indigo, leucin and tyrosin, and in many cases albumen. At the end of the first week, or at the beginning of the second week, an eruption of roseola appears, in the form of small, isolated, lenticular spots, about the size of a pin's-head, disappearing on pressure, to quickly reappear when the pressure is removed. They vary greatly in number, often from five to twenty, scattered over the lower thorax and abdomen. They may be much more numerous, several hundred in number, and may be distributed generally over the body. They may be, and indeed often are, entirely absent, especially in the milder cases. It occasionally happens that a larger, darker eruption, of a pigmentary character, appears before or with the roseola, but these have no special importance. When there has been much sweating, an abundant crop of miliary vesicles known as "sudamina" may appear on the neck, chest, and elsewhere. With the close of the second and the beginning of the third week, the typhoid symptoms develop in intensity. The stupor increases, so that the patient can hardly be roused, and is indifferent to all about him. If liquids are placed in the mouth, they are slowly swallowed. The patient lies on his back, his eyes partly closed, mouth open and black with accumulated sordes, his face is sunken, dusky, with a faint, reddish tinge in the center, the lips, now and then moving with an unintelligible muttering, are dry and cracked, and his strength is so far exhausted that he can not keep his position, but sinks toward the foot of the bed. The fæces and urine may be passed involuntarily, or the urine may be retained and dribble away, the bladder becoming enormously distended. The pulse continues frequent, from 90 to 120, or higher, but its force declines. The impulse of the heart is feeble, and hence a tendency to stasis in the lungs and brain exists. The pulse is compressible, and its tension so low that it has a double beat (dicrotic pulse). The fever of typhoid, although called continuous, is not so ; it has a distinctly remittent type. For the first week there is a gradual ascension, and, although there is a morning remission and an evening exacerbation, each exacerbation is a little higher than the preceding, until the maximum is reached. During the second week the fever is continuous ; during the third it begins to be remittent, and, during the fourth, intermittent, the daily exacerbations lessening regularly until the normal is reached. The fever at its maximum is continuous, because the daily remissions correspond to the morning and evening variations of the daily temperature in health. With the remissions at the end of the third week, there are evidences of a change for the better in favorable cases. During the third week, however, chiefly occur the complications which exercise so unfavorable an influence over the progress of the disease, but these are reserved for separate consideration. In the fourth week the patient is well aroused from the stupor, and is fully conscious of his condition. Instead of indifference, he is full of complaints. His eye is brighter, and the face, though emaciated, begins to have expression again. The delirium ceases, the nights are less disturbed, and, instead of somnolence, the sleep although refreshing is interspersed with periods of wakefulness. The tongue and gums clean, the

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* For an account of the more recent discovery of Klebs, see "Der Ileotyphus eine Schistomycose," "Archiv für experimentelle Pathologie und Pharmacologie." Zwölften Bandes, s. 231. He gives the results of examination of twenty-four cases.

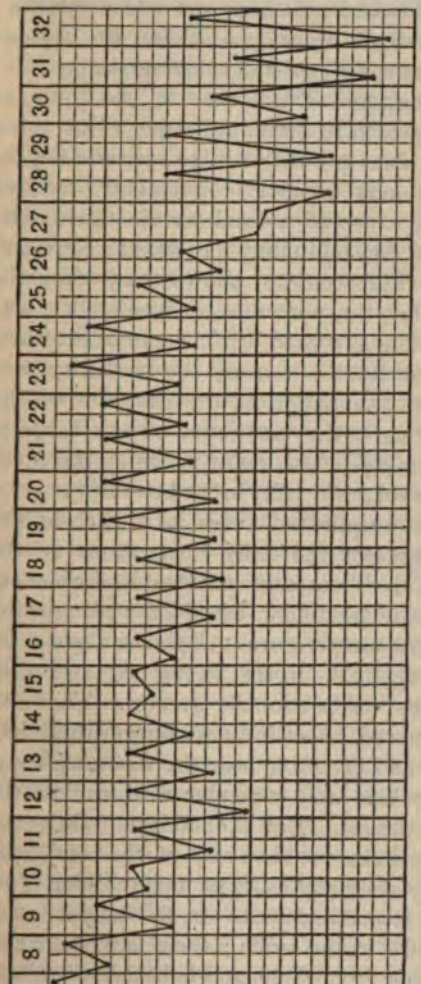

FIG. 55.—Temperature in Typhoid Fever.

appetite returns; the diarrhœa ceases, and is replaced by constipation; the flatulent distention of the abdomen subsides; the spleen shrinks; the urine becomes more abundant and limpid, and there are copious perspirations for several days, occurring especially during sleep.

Course, Duration, and Termination.—The course of the fever as described is the usual one of a perfectly developed case. But there are many variations due to individual peculiarities, to surrounding influences, to complications which may be most conveniently studied under this head. The principal cause of a fatal termination is the prolonged high temperature, and hence, in any prognostic estimate, this must be considered. Thus Liebermeister shows that, when the temperature was under 104° Fahr., the percentage of mortality was 9·6; if the temperature reached and passed 104°, the mortality was 29·1; if the temperature rose to 105·8° or over, the mortality was greater than one half. Next to the height is the duration of the fever; and, consequently, the longer the maximum of the fever is maintained, the greater the mortality. The point to which the fever attains at the end of the first week, as a rule, indicates the range of temperature to occur, for in uncomplicated cases it is then at the maximum. Furthermore, the greater the daily fluctuations of the fever, the less severe it will prove. Treatment has exercised great influence on the mortality, especially treatment based on a recognition of the importance of reducing the temperature. Age has a great influence over the termination of typhoid—in the young the mortality is proportionally less; in the aged proportionally greater. The individual constitution has an undoubted effect in increasing or diminishing the mortality; the nervous and excitable bear the disease poorly, and the phlegmatic better; the lean and muscular also endure the strain of the disease better than the fat. But the habitual indulgence in spirits has a more unfavorable influence than any of the conditions named. In every epidemic there are many cases of much milder type, and there are also irregular and abortive forms. In the milder cases, the temperature rarely exceeds 103° in the axilla; there is no delirium, only confusion of mind on awaking from sleep, and hebetude of mind; the diarrhœa is slight, and the different periods are short, so that the whole duration may be comprehended in twenty-one days. Those are regarded as abortive in which there are no prodromes, the symptoms begin abruptly, often with a distinct rigor, the temperature rising in a day or two to the maximum of 104° Fahr., and, without the weeks of continued fever, assuming the remittent and intermittent form of the fourth week at the end of the first, and terminating within two weeks. While the mild form is extremely common in this country, the abortive forms, according to the author's observation, are infrequent. The course, duration, and termination of typhoid are much influenced by the *complications*. Hæmorrhage of the intestines is one of the most important. This takes place at various times in the course of the fever, and the quantity of blood lost is very different in different cases. The blood may be pure, partly fluid and coagulated, or blackish, or converted into a tar-like mass. The second week is the most usual period for the hæmorrhage; next, the third week; but it may occur at any period. The proportion of cases of hæmorrhage to the whole number is about five per cent. When it occurs during the first week, it is a result of the increased pressure in the intestinal vessels—a necessary product of hyperæmia; if it occur in the second and third weeks, it is caused by the sloughs, a vessel being laid open by their detachment; if later, vessels are eroded by the spread of ulceration. Any considerable hæmorrhage, if no part escape externally, is announced by sudden depression, coldness of the surface, pallor, faintness, weakness of the pulse, lowering of the temperature. Unless repeated, the effect of the hæmorrhage subsides in a day or two, the pulse rises, the delirium and stupor, which may have been lessened by it, assume their former characteristics. The more severe the hæmorrhage, the more injurious. The notion has been entertained by some that a considerable hæmorrhage might have a favorable influence over the progress of a case, but the statistics are opposed to such an opinion, those of Liebermeister, for example, showing that the mortality is three times greater in those having this complication, but statistics on this point are not altogether conclusive, since usually those are the most severe cases, in other respects, in which hæmorrhage occurs. The introduction of hydrotherapy has in Germany diminished the frequency of intestinal hæmorrhage as a complication of typhoid.

Perforation, as a cause of death, occurs in from five to fifteen per cent. The period is from the third to the fifth week, although it may occur as early as the first, and is due to the extension of ulceration, the opening in the peritoneum being made at last by some hardened fæces, undigested food, sudden distention of the bowel by gas, and, it may be, by ascarides, which are often found in the peritoneal cavity afterward. The shape of the ulcer is an inverted cone, the opening in the peritoneum, the apex, having the size of a pin-head to a small pea. The ilium is the part usually perforated, but the ulcer may be situated high up in the small intestine, or it may be in the colon, especially the appendix vermiformis. Very often it is the ulcer of a solitary gland. Although the more extensive the ulcerations the greater the danger of perforation, yet it has happened that a single ulcer has opened the peritoneum. The immediate result of the perforation is shock. The surface grows cold, the temperature falls several degrees, the pulse becomes excessively feeble, and death may ensue in a condition of extreme exhaustion. Usually, however, the patient rallies, reaction ensues, and acute peritonitis rapidily develops. It sometimes happens, when the rupture may be produced by accumulation of gas, that the abdominal cavity is greatly distended by it, the epigastrium rendered prominent, and the diaphragm pushed up, impeding respiration. At the moment rupture takes place, intense pain is experienced, beginning in the right inguinal region, and radiating thence over the abdomen. The temperature rises again, after some preliminary chills, and the phenomena of peritonitis are added to the ordinary symptoms. Recovery very rarely takes place, and death occurs usually within four days after the perforation, unless, indeed, the first shock of the accident paralyzes the heart. In a few cases, with profound coma, perforation has occurred without causing any objective evidences of the complication. Perforation is much more apt to occur in men than in women. Peritonitis may be due to other causes than perforation—by the extension of ulceration to the peritoneum, by rupture of the gall-bladder, rupture of the spleen, etc. The author has met with a fatal case of rupture of the spleen, occurring during convalescence, and caused by a not violent blow on the side. Examination of the splenic region should be made with care after the second week, because of the ease with which the spleen may be ruptured. The chief complication on the part of the circulatory organs is granular degeneration of the heart-muscle already described, thromboses from cardiac weakness, forming in the heart or in the great vessels. In the respiratory system there are various changes, some of them of great importance. Epistaxis and bronchitis have been already mentioned as symptoms of the disease proper, so constant are they in appearing. Diphtheritic exudations in the fauces and ulcers of the larynx, due to diphtheritic infiltration of the mucous membrane, are occasional and very important complications. Death is sometimes unexpectedly due to œdema of the glottis, and this may be produced by a laryngeal ulcer. Atelectasis, hypostatic congestion, splenization, hæmorrhagic infarctions, and œdema, are all complications arising in the lungs from feebleness of the heart's action. Caseous pneumonia, pleurisy, and acute miliary tuberculosis are sequelæ, sometimes the outcome of the above-mentioned diseases due to stasis. Œdema of the brain is a frequent condition, which seems a necessary part of the morbid anatomy of typhoid. Besides this, there are various complications growing out of the changed state of the solids and fluids. Cerebral hæmorrhage and acute meningitis are very rare. Derangements of the mental faculties are by no means uncommon, and are due to the anæmia and the functional torpor of the gray matter. The derangement may assume the form of exaltation, or of depression and melancholy. When an hereditary tendency exists, the case assumes a higher degree of importance, those due merely to the condition of the brain, the result of the typhoid disease, recovering with less or greater promptitude. The condition of the kidneys which occurs in many cases, represented objectively by a trace of albumen in the urine, passes into well-developed Bright's disease in a small proportion of them. These go through the usual course, and terminate in recovery. Hæmorrhagic infarction occurs in a few cases. The menses frequently appear during the course of typhoid, and exercise a rather favorable influence over the course of the disease. Abortion is apt to occur, and of course adds to the gravity of the situation. On the part of the skin, the most important complication is that of *bed-sores.* The parts subjected to pressure are those which slough—the sacrum, nates, great trochanters, and the crest of the ilium. In some subjects, so depraved is the condition of the solids, that any part subjected to pressure sloughs. The depth and extent of the sloughing vary from redness, inflammation, and abrasion of the skin, to destruction of the skin, fascia, and muscles, extending to the periosteum. The effect of this complication depends on the extent of the injury done. When there is considerable sloughing, suppuration, and decomposition, fever will be excited, and systemic infection, septicæmia, and pyæmia result. Falling out of the hair and arrest of the growth of the nails are usual complications.

Relapses.—Increased fever, due to some complication, may be confounded with a genuine relapse, but the latter pursues the ordinary course of the fever, except that it is more rapid in its course and shorter in its duration. There occurs in the relapse a similar range of temperature, the spleen enlarges, roseola appears, and the other symptoms in their order come on. Of itself the relapse is milder, but the subject enduring it is enfeebled by an illness, so that the danger must be regarded as greater. The number of cases undergoing relapse varies from six to twelve per cent.

Treatment.—Although for typhoid, a specific disease, we have no specific remedy, a treatment has originated in Germany which is known as the specific treatment. Mercury and iodine are the specific remedies. There is no doubt, if statistics may be depended on, that calomel, in large doses during the first week, favorably modifies the disease. Ten grains in a single dose, on alternate days, is about the average of the quantity given by various therapeutists. If the temperature is high, it may be given on successive days, but the danger of inducing salivation is great, when it is administered at short intervals. The effect of the mercurial treatment is to lower the temperature, to diminish the severity, and apparently lessen the duration of the case. The treatment by iodine consists in the administration of Lugol's solution—from three to five minims in water three times a day, and continued during the first two weeks certainly, and probably up to the beginning of convalescence. Taking the figures of Liebermeister for illustration, they show that while the mortality under ordinary treatment reached 13·2, under calomel it was 8·8, and under iodine 10·9. The author's experience is, that the administration of iodine has a favorable effect on the course of the disease. He has used, with apparently decided success, the combination of iodine and carbolic acid (℞ Tinct. iodinii ʒ ij, acid. carbolic. ʒ j. M. Sig.

abdominal cavity is greatly distended by it, the epigastrium rendered prominent, and the diaphragm pushed up, impeding respiration. At the moment rupture takes place, intense pain is experienced, beginning in the right inguinal region, and radiating thence over the abdomen. The temperature rises again, after some preliminary chills, and the phenomena of peritonitis are added to the ordinary symptoms. Recovery very rarely takes place, and death occurs usually within four days after the perforation, unless, indeed, the first shock of the accident paralyzes the heart. In a few cases, with profound coma, perforation has occurred without causing any objective evidences of the complication. Perforation is much more apt to occur in men than in women. Peritonitis may be due to other causes than perforation—by the extension of ulceration to the peritoneum, by rupture of the gall-bladder, rupture of the spleen, etc. The author has met with a fatal case of rupture of the spleen, occurring during convalescence, and caused by a not violent blow on the side. Examination of the splenic region should be made with care after the second week, because of the ease with which the spleen may be ruptured. The chief complication on the part of the circulatory organs is granular degeneration of the heart-muscle already described, thromboses from cardiac weakness, forming in the heart or in the great vessels. In the respiratory system there are various changes, some of them of great importance. Epistaxis and bronchitis have been already mentioned as symptoms of the disease proper, so constant are they in appearing. Diphtheritic exudations in the fauces and ulcers of the larynx, due to diphtheritic infiltration of the mucous membrane, are occasional and very important complications. Death is sometimes unexpectedly due to œdema of the glottis, and this may be produced by a laryngeal ulcer. Atelectasis, hypostatic congestion, splenization, hæmorrhagic infarctions, and œdema, are all complications arising in the lungs from feebleness of the heart's action. Caseous pneumonia, pleurisy, and acute miliary tuberculosis are sequelæ, sometimes the outcome of the above-mentioned diseases due to stasis. Œdema of the brain is a frequent condition, which seems a necessary part of the morbid anatomy of typhoid. Besides this, there are various complications growing out of the changed state of the solids and fluids. Cerebral hæmorrhage and acute meningitis are very rare. Derangements of the mental faculties are by no means uncommon, and are due to the anæmia and the functional torpor of the gray matter. The derangement may assume the form of exaltation, or of depression and melancholy. When an hereditary tendency exists, the case assumes a higher degree of importance, those due merely to the condition of the brain, the result of the typhoid disease, recovering with less or greater promptitude. The condition of the kidneys which occurs in many cases, represented objectively by a trace of albumen in the urine, passes into well-developed Bright's disease in a small proportion of them. These go through the usual course, and terminate in recovery. Hæmorrhagic infarction occurs in a few cases. The menses frequently appear during the course of typhoid, and exercise a rather favorable influence over the course of the disease. Abortion is apt to occur, and of course adds to the gravity of the situation. On the part of the skin, the most important complication is that of *bed-sores.* The parts subjected to pressure are those which slough—the sacrum, nates, great trochanters, and the crest of the ilium. In some subjects, so depraved is the condition of the solids, that any part subjected to pressure sloughs. The depth and extent of the sloughing vary from redness, inflammation, and abrasion of the skin, to destruction of the skin, fascia, and muscles, extending to the periosteum. The effect of this complication depends on the extent of the injury done. When there is considerable sloughing, suppuration, and decomposition, fever will be excited, and systemic infection, septicæmia, and pyæmia result. Falling out of the hair and arrest of the growth of the nails are usual complications.

Relapses.—Increased fever, due to some complication, may be confounded with a genuine relapse, but the latter pursues the ordinary course of the fever, except that it is more rapid in its course and shorter in its duration. There occurs in the relapse a similar range of temperature, the spleen enlarges, roseola appears, and the other symptoms in their order come on. Of itself the relapse is milder, but the subject enduring it is enfeebled by an illness, so that the danger must be regarded as greater. The number of cases undergoing relapse varies from six to twelve per cent.

Treatment.—Although for typhoid, a specific disease, we have no specific remedy, a treatment has originated in Germany which is known as the specific treatment. Mercury and iodine are the specific remedies. There is no doubt, if statistics may be depended on, that calomel, in large doses during the first week, favorably modifies the disease. Ten grains in a single dose, on alternate days, is about the average of the quantity given by various therapeutists. If the temperature is high, it may be given on successive days, but the danger of inducing salivation is great, when it is administered at short intervals. The effect of the mercurial treatment is to lower the temperature, to diminish the severity, and apparently lessen the duration of the case. The treatment by iodine consists in the administration of Lugol's solution—from three to five minims in water three times a day, and continued during the first two weeks certainly, and probably up to the beginning of convalescence. Taking the figures of Liebermeister for illustration, they show that while the mortality under ordinary treatment reached 13·2, under calomel it was 8·8, and under iodine 10·9. The author's experience is, that the administration of iodine has a favorable effect on the course of the disease. He has used, with apparently decided success, the combination of iodine and carbolic acid (℞ Tinct. iodinii ʒ ij, acid. carbolic. ʒ j. M. Sig.

One to three drops three times a day). Nitrate of silver, sulphate of copper, arsenic, and turpentine, each has an advocate of its usefulness—all being directed against the intestinal complication or lesion. As, however, the main point in the management of typhoid is to keep the temperature within safe limits, the treatment directed to that end is the most important. The antipyretics available for this purpose are hydrotherapy, quinine, antipyrin, the salicylates, benzoates, etc. The method of hydrotherapy consists in immersion in water at a certain temperature, the wet pack, and local abstraction of heat by special appliances. As private houses are unprovided with the means of administering baths to fever-patients, this method can be utilized only in hospitals. The method of gradual reduction of heat we hold to be preferable. The patient is put in the water at 98°, and then by the addition of cold water the temperature of the bath is brought down to 60° Fahr. The thermometer must be constantly in position to observe the effect, and the duration of the bath ought not to exceed ten to fifteen minutes. The temperature requiring the bath is any considerable elevation above 103° Fahr. (axillary), and the repetition of it is determined by the effect—every two to every six hours, night as well as day, may be regarded as usual. If the patient is made faint or depressed, some stimulant should be given before, during, or subsequent to the bath, according to the result. If the bath is impracticable, the wet pack may be used with equal effect. The bed is protected by a gum cloth ; a sheet is wrung out of cold water ; the patient is thoroughly wrapped in it, and then covered up with blankets for a few minutes, when the process is renewed if necessary. The same rules hold good with regard to the repetition and management of the pack as of the bath, and the results achieved are equally beneficial. The temperature of the body may also be reduced by ice-bags applied to the abdomen, and by ice-water injections in the rectum, but these latter can not be utilized in typhoid. There are several contra-indications to the use of cold baths. The first and most important is hæmorrhage from the intestines, the next is great weakness of the heart's action, and the third is coldness of the surface with high internal heat. Next to hydrotherapy, and probably superior as a remedy for reducing abnormal temperature of the body, is quinine. Notwithstanding the good results which have been obtained from baths, it is probable that quinine will always be preferred by many, because of the readiness with which it may be brought to bear on the production of heat. Indeed, Liebermeister, a strong advocate for hydrotherapy, says, if he "were forced to the unpleasant alternative of adopting only one or the other of these two means—cold water or quinine—I should, in the majority of cases, choose the latter." To reduce the abnormal temperature, antipyretic doses are required, from twenty to forty grains. A decline of several degrees, and lasting a number of hours, will be caused by a sufficient dose, and a less effect than this will not justify the employment of the remedy. It is a good plan to prescribe a scruple every four hours, until a decided reduction of temperature takes place, then its use should be suspended until the temperature begins to rise again.

After extended observation of the practical results of the antipyretic method, as carried out by the administration of the remedies belonging to this group, the author has come to the following conclusions: If the temperature at the maximum of the daily curve does not exceed 103° Fahr., it is better not to interfere by an attempt to lower the body-heat. If the temperature persistently rises above this point, antipyretics least perturbating should be administered at the period of remission, and in sufficient quantity to make the necessary impression, when they should be discontinued until an exacerbation is threatened. Antipyrin, disturbing least the digestive organs, should be preferred after quinine, or to quinine if this remedy fail to have the desired effect. It is not good practice to give quinine in so-called tonic doses (two to four grains) for the following reasons: It disorders the stomach; it increases the headache and the gloom, the hebetude of mind and the muttering delirium, and thus contributes to the wakefulness. There is no benefit derived from it to compensate for these considerable disadvantages.

If, in the treatment of typhoid, the temperature be prevented rising beyond safe limits, there will be less and less need for attention to complications. Nevertheless, we must be prepared to obviate the tendency to death, and to correct complications. Failure of the heart requires stimulants, but otherwise stimulants should not be given in typhoid, except in the case of those addicted to their use, who require a regulated daily amount. Restlessness and prolonged wakefulness are as a rule most successfully relieved by morphine and belladonna. Chloral must be used with caution, because of the weakness of the heart-muscle. If the tongue is dry, if there is great thirst, and the abdomen is much distended with gas, turpentine is highly useful. Muriatic acid also acts well under the same circumstances. If the bowels act too freely, nitrate of silver, with a little opium, Fowler's solution and laudanum, bismuth and carbolic acid, especially the last-named combination, will check them sufficiently. Two or three stools a day are not interfered with, unless copious and exhausting. Careful alimentation best regulates the bowels. If hæmorrhage occur, intestinal movements must be suspended by opium, the flow of blood controlled by ergotin hypodermatically and ice to the abdomen. Tannin, alum, and solution of chloride of iron may be prescribed internally. If perforation occur, opium, especially morphine, hypodermatically, is our one remedy. Stimulants may be given cautiously, and absolute rest should be maintained. Bed-sores are best managed by cold-water bags and the removal of pressure. Before the skin breaks, it should be frequently washed with alcohol and Goulard's extract to harden it. The best dressing for a bed-sore is a mixture of equal parts of copaiba and castor-oil. A large yeast-poultice is an excellent application, especially when more or less weight is still borne by the sore surface. The alimentary treatment of typhoid fever is very important. The principal lesions being in the intestinal canal, the diet must be arranged accordingly. Dr. George Johnson has shown us that many cases of typhoid need nothing more than rest in bed and milk diet; and Sir William Jenner has pointed out how useful milk is, and how injudiciously it is given in many cases. We learn from these able physicians, that milk is peculiarly adapted to serve as the food for typhoid-fever patients, but that it must be given in moderate quantity, and at suitable intervals. Milk should be administered about every three hours, and from two to four ounces at one time. Or milk may be given in alternation with a little weak mutton, beef, or chicken broth. If milk is not borne well, it may be diluted with barley-water. A little of Scheffer's pepsin solution and muriatic acid ought to be administered immediately after the aliment, if it is rejected by vomiting or passes by stool unchanged. Beef-essence, as usually prepared and given to typhoid-fever patients, is very difficult of digestion, acts as a laxative, and may be seen in the evacuations precisely in the state in which it was swallowed. As the adynamia increases, egg-nogg, fortified by whisky or brandy, comes to be a most useful aliment, of which the patient may partake freely, but at regular intervals. Sufficient time ought to be allowed for the aliment given at one time to be digested, before another supply is turned into the stomach. A moderate quantity of a light wine should be allowed during the first two weeks, and whisky and brandy given in egg-nogg or milk-punch the third and fourth weeks. Half an ounce to an ounce of wine and a half-ounce of whisky or brandy need rarely be exceeded at one time, nor more frequently than once in three hours, unless there be a special requirement. Mild cases need no stimulant. The dejections of a typhoid patient should be at once disinfected by a strong solution of sulphate of iron or chloride of zinc. The patient's bed should be free from all unnecessary appendages, and be placed in the middle of the apartment. Air should be freely admitted. But one person should, as a rule, be permitted in the apartment at a time, and the patient's attention should not be attracted to persons and things about him.

TYPHO-MALARIAL FEVER.

History.—This term was invented by the late Dr. Woodward, of the Army, to express his conception of a hybrid formed by a combination of typhoid fever and malarial fever. As it was used in the medical reports of the army, and hence at once adopted by a large proportion of the physicians of the United States, it came to be regarded as signifying a new disease. This unfortunate misconception was further promoted by Dr. Woodward's published utterances on the subject. In his work on "Camp Diseases" we find him declaring that typho-malarial fever is a substantive malady, that has a special morbid anatomy. He found, as he allĕged, anatomical changes belonging to and characteristic of the new disease. The author of this work was the only one to oppose these doctrines and Dr. Woodward's typho-malarial fever becoming a fixture in the nomenclature of diseases. The author maintained that Dr. Woodward had committed an error of observation, that the morbid anatomy of typhoid was in nowise altered by an existing malarial complication, and that, symptomatically, remittent fever running into a typhoid state was mistaken for true typhoid. The author further maintained that, admitting the existence of a malarial complication, this no more constituted a reason for constructing a new disease than the presence of such other complications as scurvy, crowd-poisoning, rendered the manufacture of new terms necessary to express these relationships. Dr. Woodward, after ten years' silence, at last, in a paper read before the International Medical Congress at Philadelphia, retracted his former statements, and admitted that the morbid anatomy of typhoid fever remained unaltered by any malarial complication. Consequently, his assumption of a typho-malarial fever fell unsupported. Unfortunately, an error of this kind is more easily established than it is destroyed by subsequent refutation. The reviewers of this work, who have only seen that the author did not admit the existence of a typho-malarial fever, and criticised the omission in an unfavorable tone, seem to be totally ignorant of the fact that the author of the term admitted its inapplicability and the error of his observations.

Clinical Aspects of Malarial Typhoid.—Although typhoid fever continues to be typhoid, practical physicians have long known that, when the typhoid germ develops in an organism saturated with the malarial poison, the clinical features of the fever are somewhat modified; but the modification consists in the increased excursions of the daily temperature. Influenced in their conception of typhoid as a continued fever by this term, physicians have too often overlooked the fact that the thermal line of typhoid is that of a remittent fever, and hence the daily remission and exacerbation were regarded as the expression of the malarial infection. It is merely in some increase of the sweep of the thermal wave that the influence of the malarial complication is made evident. After the typhoid process has expended itself, and in a perfectly legitimate manner, the malarial element begins a disturbance. Then it is that a remittent, or more frequently a quotidian or tertian intermittent, comes on to interrupt and prolong the stage of convalescence.

The Role of Quinine in Malarial Typhoid.—If any additional evidence were needed to prove the falseness of the conception which regards the so-called typho-malarial fever as a morbid entity, it is afforded us in the behavior of quinine. All the world knows that quinine manifests no more power to control this than to arrest typhoid. There comes a period, however, when the use of quinine is indispensable. It is when, after the exhaustion of the typhoid process, the malarial element initiates a characteristic disturbance on its own account. Then, by its timely exhibition, a convalescence that might have been protracted and unsatisfactory is rendered shorter and milder.

The Typhoid State in Remittent Fever.—It is necessary to note another source of error in regard to typho-malarial fever. Cases of remittent fever of severe form, if not arrested by massive doses of quinine, will assume more and more nearly a continued type, and will finally pass into a typhoid state. Very often, no doubt, this typhoid condition is confounded with typhoid fever, but a study of the morbid anatomy will disclose the error.

TYPHUS FEVER.

Definition.—A febrile affection, self-limited, and characterized by profound adynamia, a peculiar petechial eruption, favorable cases terminating by crisis at the end of the second week. Typhoid and typhus are now almost universally regarded as distinct affections. Stokes,[*] however, takes a different position, and maintains that the points of resemblance are greater than the differences.

Causes.—As a rule, typhus prevails in seaport towns, where it is known as "ship-fever"; but it has under some circumstances ravaged continents, as during the great famine periods: Ireland has been decimated, and, under similar circumstances, Italy and Austro-Hungary have been severely visited.[†] Typhus now prevails in crowded ships, asylums, and jails—where great numbers are accumulated together, are depressed by poor food and bad air. It is seen in this country only at our seaport towns, and the author's personal experience is limited to cases observed at the Baltimore Infirmary, admitted to the service of the late Professors Power and Chew from ships in the harbor in the years 1850–'53. How evil soever may be the hygienic influences, typhus does not originate spontaneously; the peculiar germ must be introduced from without. Of the nature, form, and condition of the germ we know nothing. The disease is contagious, and the contagious principle increases in virulence the more crowded and numerous the patients within a given area, and the more unfavorable the hygienic influences and the bodily state of those attacked. Hence the terrible force of the poison during the famine periods in Ireland. The disease is more frequent among males than among females, and occurs by preference during the most active period of life, or from fifteen to

[*] "Lectures on Fever," London, Longmans, Green & Co., 1874, p. 86.

[†] "Traité de Climatologie Médicale," tome iv, p. 362, *et seq.*

fifty. Like other acute infectious diseases, one attack serves to exempt from future attacks.

Pathological Anatomy.—We do not find in typhus the definite series of changes which so individualize typhoid. The solids and fluids generally are deeply injured. Vascular turgescence is noted in the upper part of the small intestines and the ileum. In the midst of stellate or arborescent injection in the ileum, there are sometimes small spots of ulceration, not all like the ulcerations of typhoid, and occurring in only five per cent. of the cases.* Changes—thickening and deposits—in the mesenteric glands are very uncommon. More or less congestion of the spleen, liver, and kidneys, with granular degeneration more or less advanced, is noted in a portion of the cases. A similar change—granular degeneration—occurs in the heart as well. There is present some serum in the sac of the pericardium. The blood is dark, fluid, and not firmly coagulable, but thrombi are found adherent to the walls of the large veins. There is more or less fluid in the subarachnoid spaces, and the membranes and cerebral substance are more or less injected. The mucous membrane of the bronchi are hyperæmic and sometimes inflamed, and occasionally atelectasis and pneumonia are encountered. The muscles present the changes of granular degeneration.

Symptoms.—There may or may not be a prodromic stage, and, when it does occur, it is of short duration. The patient is dull, heavy, dispirited, experiences a strong sense of fatigue, has headache, is restless and wakeful at night. In a few days the effort to keep up is abandoned, and the patient betakes himself to bed, thoroughly exhausted. In other cases, of which the great Irish epidemics have furnished numerous examples, the patient is suddenly seized, and passes at once into a state of profound adynamia, or he walks to the hospital, is put to bed, and in twenty-four hours he lies helpless, comatose, and sinking. There may be a slight chill at the onset, or nausea and vomiting may inaugurate the symptoms. A very severe headache and pains in the back and limbs are now experienced. The head feels hot ; there is much giddiness when the attempt is made to rise ; and sneezing, with other symptoms of catarrh, and noises in the ears are also experienced. The fever rises rapidly from the beginning, the pulse ranges from 90 to 120 at once, and the temperature by the third or fourth day has attained to 103° or 104° Fahr. in the morning and 105° or 106° in the evening. Again, it sometimes happens, so profound is the intoxication, that the forces are inadequate to maintain the pulse at or above normal and the temperature above 99°. There may be high temperature temporarily without any special significance ; but persistently high temperature bodes ill. Extreme weakness and a deep, apathetic listlessness soon come on, when the patient lies on his back, oblivious to all about him ; his eyes are half closed, and are dull and glazed ; his mouth is half open, the lips dry and cracked, the teeth covered with sordes ; his face is dusky, which is the general tint of the skin, and the malar protuberance has a reddish-brown color. Toward the end of the first week the characteristic eruption of typhus makes its appearance on the back between the scapulæ in males, on the chest and abdomen in females, and spreads thence over the rest of the body. They are a half line to a line in diameter, reddish-brown in color, a little elevated above the general surface, disappearing on pressure, to reappear when the pressure is removed. They may be very numerous, so that a dozen will be contained in a square inch, or they may be sparse and larger in size. Successive crops appear, and the duration of the eruption stage is from five to seven days, so that it may be expected to disappear from the twelfth to the fourteenth day. Prognostications may be drawn from the appearance of the eruption. If it is rose-colored, the general tint of the skin being good, the condition is favorable ; if a dusky-brown, rather livid color, the skin also dusky, the condition is unfavorable. Trousseau* formulates the significance of the eruption as follows : "The gravity and duration of the malady are in relation to the abundance and depth of color of the eruption." Besides the measles-like eruption, which is characteristic, there are in some epidemics spots and patches of purpura, of varying size, and the larger extravasations known as vibices. Both of these indicate a low form of the disease, and are, therefore, symptoms of evil augury. Sudamina also occur, but these have no special significance, unless differing from ordinary sudamina in the character of their contents, which, if bloody, or having a putrescent odor, show a bad state of the tissues. At the close of the first or beginning of the second week, instead of there being a merely clouded state of the mental faculties, active delirium may ensue. It may be very violent, the patient difficult of control, striking and fighting all who approach, trying to get out of bed, etc. This condition, which has been happily designated *delirium ferox*, may continue for days and nights, the patient sleeping none, there being at the same time intense fever, rapid action of the heart, injected conjunctivæ, great intolerance of light, and contraction of the pupils. But this active and violent delirium is much less common than low-muttering delirium in which the illusions and hallucinations form the topics of the unintelligible rambling. The patient usually lies in an entirely passive state, taking food mechanically, sleeping but little, although in a constant soporose state, the pulse ranging from 120 to 140, double-beating, easily compressible, the surface of the body presenting a dusky, cyanosed appearance, and the actual condition being that of profound and increasing prostration. There is usually some dry cough. The bowels are at first rather confined, and during the height of the disease the dejections are scanty, rather infrequent, but consist of somewhat loose, offensive, dark stools. There is no distention of the abdomen. The spleen is enlarged, and can be made out projecting downward. The urine is scanty, high-colored, specific gravity high, and usually contains albumen. During the stupor, urine and fæces are passed involuntarily. A very peculiar and distinctive odor is maintained by many to exist. Trousseau regards it as *sui generis;* but we believe it to be similar to that which is to be detected about all fever-patients so oblivious to their natural wants. During the second week the prostration is so profound that patients die, without any special complication, from failure of the heart. The temperature of the skin falls; the purpuric spots enlarge; parts exposed to pressure—the sacrum especially—soften and ulcerate; the pulse becomes small and irregular; the impulse of the heart is scarcely perceptible, and the first sound is no longer audible. In this condition the patient may remain for a day or two, even longer, suspended between life and death—the stupor may deepen into fatal coma, or death may be induced by sudden engorgement of the lungs, or the heart fails, the pulse becomes imperceptible at the wrist, and the surface cold, and covered with a cold sweat. Instead of a fatal termination, a large proportion recover. About the fourteenth day, if a change for the better is to occur, phenomena of a rather critical character supervene. The patient falls into a quiet sleep lasting several hours, and he awakes refreshed, and with consciousness restored, but oblivious of all that has transpired, and feeling an extreme degree of feebleness. The pulse lessens in frequency, but gains in volume; the tongue begins to clean and is moist; the skin is covered with a warm perspiration, and a little appetite is felt. The critical phenomena which may accompany this change for the better consist of a free sweat, a diarrhœa, or an abundant urinary discharge, with large deposits (Murchison[*]).

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* Lyons, *op. cit.*, p. 142, *et seq.*

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* "Clinique Médicale," tome i, p. 299.

Course, Duration, and Termination.—There are great variations in the course of cases of typhus during the epidemics. In the mildest cases the pulse may not exceed 100, the tongue may never become dry and brown, there may be only temporary confusion of mind, and somewhat troubled sleep. There are extreme cases, in which the patient is stricken down with the intensity of the poison, and at once passes into a state of profound prostration, with disorganization of the blood; and, without any complication to account for it, life is extinguished in a few days after the onset of the disease. Usually, however, the fatal result may be referred to the rise of some complication. Some of the most important are the pulmonary: bronchitis, hypostasis, pneumonia, gangrene of the lung, and pleurisy. During the course of typhus, frequent examinations should be made of the thoracic organs, since the insensibility is so profound that the patient may not present any indications of the complications. Especially should an increased rapidity of breathing become manifest, or the alæ of the nose labor, or the lividity of the face deepen, attention should at once be directed to the state of the thoracic organs. The most usual of the thoracic complications is bronchitis, and it is not always shown by cough, but only by moist *râles*. The danger consists in an extension to the smaller tubes, and the association of hypostatic congestion with capillary bronchitis. When the adynamia is very deep, the tubes may become paretic, and can not expel the accumulating mucus, death occurring in asphyxia. The association of hypostatic congestion with bronchitis is the most usual cause of death in typhus, taking the general order of cases. Pneumonia is uncommon, but gangrene is comparatively frequent in famine-typhus. Thrombosis of the femoral artery sometimes occurs, but the chief complications on the part of the blood are those due to its disorganization: purpuric spots, hæmorrhages by the nose, bronchial tubes, stomach, intestines, and kidneys, and a more or less extensive general cyanosis. Imbecility and mania are sometimes sequences of typhus, but there are complications of a paralytic kind occurring during the course of the fever, or during convalescence, such as hemiplegia, paraplegia, or affections of the special senses, amaurosis, and especially deafness. These are usually temporary, and due to the extreme degree of anæmia produced by the fever, but some of them are more permanent, as the deafness due to suppuration of the middle ear. Complications on the part of the skin are often very severe, notably the extensive bed-sores, gangrene of the skin, and furuncles. A whole extremity may become gangrenous. Erysipelas of the scalp and face, suppuration of the parotid gland, and buboes, are also encountered. All of these complications increase the gravity of the case, and in proportion to their importance. The duration is also more or less influenced by the complications. The ordinary duration of a mild, uncomplicated case is about three weeks. The Germans recognize an abortive form of typhus, terminating by crisis about the seventh day, but such cases, it seems to the author, belong to a different order. A case of typhus may be protracted by complications four, five, or six weeks. Even in the severer epidemics the majority recover. Much depends on the type of the cases. Those characterized by intense fever and active delirium are called *inflammatory;* those in which the merely nervous symptoms, as delirium, stupor, subsultus tendinum, predominate, are designated *ataxic;* and those in which a profound prostration comes on are known as *adynamic* (Murchison). In the severe epidemics which have visited Ireland and India one fifth have proved fatal, and this was the mortality at the London Fever Hospital for fourteen years. In some epidemics, the mortality has risen to forty per cent., and even higher, and in others has fallen to eight per cent. The type of the epidemic, as well as of individual cases, is, therefore, a large factor in determining the mortality. The mean mortality is from fifteen to twenty per cent. The disease is more fatal in males than in females, and is less fatal in childhood, the mortality increasing with age.

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[*] Murchison "On Fevers," *op. cit.*

Diagnosis.—Stokes is the only author of any prominence advocating the identity of typhoid and typhus. The prodromic stage is more usual and protracted in typhoid; the onset of stupor and delirium is earlier and more pronounced in typhus; in typhoid there are meteorism, gurgling in the right iliac fossa, and diarrhœa—in typhus these are wanting; in typhoid there is a roseola eruption of a small number of spots; in typhus there is a petechial eruption, which is abundant over the body; the duration of typhus without complications is about two weeks, often terminating with crisis—of typhoid, four weeks, by slow decline of fever; on *post-mortem* examination, thickening and ulceration of Peyer's patches and of the solitary glands and enlargement and softening of the mesenteric glands are seen in typhoid, while no similar or corresponding changes take place in typhus.

Treatment.—The same means of treatment pursued in typhoid are equally applicable here, except that the adynamic condition appears sooner, and is more profound, requiring a somewhat earlier resort to stimulants. The alimentation should be carefully prescribed from the beginning, and should consist of milk, eggs, animal broths, and a moderate quantity of wine, which should be changed to whisky or brandy as the prostration increases. Still more than in typhoid is it necessary in typhus to keep the temperature within safe limits by the use of antipyretics. Cold baths, or the wet pack, quinine, and digitalis, are used as in the treatment of typhoid, under the same rules and regulations. As certain critical phenomena may ensue at or about the end of the second week, it is important to be prepared for them, lest the revolution which then takes places may tax too heavily the vital resources. As typhus is distinctly contagious, isolation of the patient is demanded by every consideration, and all of the patient's excretions should be disinfected and removed without delay.

RELAPSING FEVER.

Definition.—This is an acute, infectious, febrile disease, self-limited, and characterized by the occurrence of a febrile paroxysm, lasting about one week, succeeded by an entire intermission of four or five days' duration, which is in turn followed by a relapse like the first seizure, although shorter.

Causes.—Relapsing fever is a distinctly contagious affection. Some excellent illustrations of the modes in which it may be communicated have been narrated by Parry,* and every epidemic furnishes examples. The poison acquires the greater activity the more filthy, crowded, and unhealthy the population amid which it prevails. The larger the number of sick, ill with the disease, crowded into a given locality, and the more unhygienic the local conditions about the sick, the more virulent becomes the poison. Articles of clothing which have been about the sick will retain the contagious principle for a long time, and those who have been in the presence of the sick can convey the poison to the healthy at a distance. It seems in a high degree probable that drinking-water may be contaminated and spread the poison. So rapidly are members of a family attacked, after one case has been introduced, that some general cause might be supposed to act on all simultaneously. The disease attacks by preference the young, the liability lessening after thirty, and apparently ceasing after fifty. In this disease we seem nearer than in almost any other to a correct knowledge of the nature of the morbific principle, since the discovery by Obermeier in 1873 of a minute organism in the blood of relapsing-fever patients. Unlike most of the other fevers, the occurrence of one attack of relapsing fever does not purchase an immunity against subsequent attacks; indeed, the liability to it seems rather increased by previous attacks. An intimate relation apparently exists between relapsing fever and typhus, for Lebert has ascertained that, of fifty-three cases of relapsing fever, all were attacked with typhus within a few weeks to several months. Although the natural home of relapsing fever is Ireland, it has spread over England, on to the Continent, and has reached this country, distinct epidemics having occurred since 1850 in New York, Philadelphia, and other cities. It occurs at all seasons.

Pathological Anatomy.—The alterations produced by relapsing fever are by no means characteristic. During life minute organisms are found in the blood, but, according to Lebert,† "they were searched for in vain in the spleen, lungs, and other organs." During the primary attack and relapse these organisms are present, but they disappear, or usually do, during the period of intermission. These bodies consist of minute spiral filaments, constantly in motion. They never exceed 0·001 mm. in diameter, and 0·15 to 0·2 mm. in length (Lebert). The very lively, twisting, and elongating motions of these spiral bodies cease as the blood coagulates, and those observed in the serum of the blood are often embraced in a granular substance, probably albuminous.‡ The relative proportion of white blood-corpuscles is increased. The spleen is usually considerably enlarged, and may be either firm or soft. "Miliary aggregations of a dull-yellow color, and containing granular detritus, with occasionally cell-elements and free nuclei," are found in the spleen in some cases, and in other cases "wedge-shaped infarctions." These may be supposed to have their origin in embolisms formed by masses of the spiral organism. The liver is also somewhat enlarged, and the acini are in many instances pale and clouded; and there are, rarely, it must be admitted, minute deposits like those mentioned as present in the spleen. The gall-bladder is full. The kidneys, like the liver and spleen, are somewhat swollen; the cortex is pale, and cloudy swelling and granular infiltration are to be seen in the tubules. In the intestinal canal some thickening of the solitary glands and patches of Peyer occurs, also in the mesenteric glands; but these changes are trivial as compared with those of typhoid fever. Sometimes in various parts of the mucous membranes minute extravasations of blood are found. The only change in the heart is a granular condition of its muscular tissue, such as occurs in febrile affections, and a similar change is to be seen in the muscles, generally due to the same cause.

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* Dr. J. S. Parry, "The American Journal of the Medical Sciences," October, 1870.

† Ziemssen's "Cyclopædia," vol. i, *op. cit.*

‡ Dr. Paul Guttmann ("Verhandlungen der physiologische Gesellschaft zu Berlin," No. 7, 1880) has examined the blood of more than two hundred cases of relapsing fever, and finds the characteristic spirilli of Obermeier only during the pyretic period. These

Symptoms.—From the period of exposure, or of reception of the morbific material, until the first phenomena of the disease are manifest—the incubation—about five to seven days elapse. This is not invariable, and must therefore be regarded as a close approximation only. There is no real prodromic period. Just as the disease is about to appear the patient experiences a general *malaise*—some pains in the head and limbs, wakefulness, loss of appetite, etc. The malady begins rather abruptly with fever, in only one half of the cases is there chilliness, and in a much smaller number a distinct rigor. In some epidemics there are irregular chills, and occasional sweats for the first two or three days, simulating an intermittent fever. In many cases the fever is high and the symptoms severe from the beginning; in other cases the patient keeps about for the first few days. With the initial fever there are usually nausea and vomiting, and, if not in the beginning, in a very short time there is a marked degree of debility. The fever is of the remittent type, with a morning remission and an evening exacerbation—the morning temperature being at 102° to 103° Fahr., and the evening temperature at 104° to 105°. The pulse corresponds, ranging from 110 in the morning to 130 in the evening, and is rather weak, usually dicrotic, or wanting in tension. The tongue is coated and soon becomes very dry and sore; the bowels are constipated. The chief source of suffering at the outset is the pain in the back and limbs, but all the muscles of the body soon become the seat

new observations confirm what is stated in the text. Dr. Guttmann further shows that the spirilli are genuine parasites. (See also Virchow's "Archiv," Band lxxx, s. i, 1880.)

of very violent grinding, piercing, lancinating pains, and these pains are increased by movement or pressure. The most aggravated of these pains are those felt in the calf of the leg. The headache, which was so pronounced in the beginning, lessens somewhat in severity as the muscular pains develop. About the second day a painful sense of weight and pressure is experienced in the right and left hypochondrium, especially in the left, and is caused by enlargement, with congestion, of the liver and spleen. The spleen especially enlarges very considerably, projecting below the ribs. The area of hepatic dullness is also much increased, and the margin of the liver can be felt several fingers' breadths beyond the ribs. This increase in the dimensions of these organs begins on the second day, and increases day by day, to diminish during the interval or intermission. Besides the increase in volume, these organs become very sensitive to pressure, and continue tender as long as they are enlarged. There is no tympanitic distention of the abdomen, no diarrhœa, no rose-spots, but more or less vomiting persists during several days, the vomited matters consisting of a greenish, acid fluid. There is no delirium, the nights are much disturbed by pain, but the mind is unclouded. The urine frequently contains albumen, but its composition in other respects is that of the urine of febrile diseases in general. More or less sweating occurs, but no amelioration of the fever is produced, for the skin continues hot, while there is a general moisture of the surface. The fever, the pains, the nausea and vomiting, the tumefaction of the liver and spleen, continue up to the end of the paroxysm. It is not surprising that, under these circumstances, there should be weakness and emaciation. In a small proportion of cases jaundice appears at some period during the first paroxysm. Toward the end of the first week, on the fifth, sixth, or seventh day, all of the symptoms attain their maximum and the case looks truly formidable, when a sudden defervescence takes place, and with it a remarkable diminution in all of the symptoms. Profuse sweating sets in, and the temperature falls to normal and below, a variation of five or six degrees taking place from night to morning. The pulse also descends from the high point at which it had been at the maximum, to the normal, or even below. Corresponding changes ensue in the other symptoms. A feeling of comparative comfort is substituted for the severe pains; appetite replaces nausea or disgust for food; the bowels act normally; the swelling and tenderness of the liver and spleen disappear, and the jaundice, if present, begins to fade; the tongue clears off; sleep is restored, and the strength gains rapidly, so that in a day the patient is disposed to get up and regards himself as well, although somewhat weak. The improvement continues, and hence it is a matter of extreme surprise to the patient, if unfamiliar with the nature of the malady, to be attacked with a relapse. The period of intermission is not a fixed period, and varies from four days to one week, very rarely to two weeks. Complete recovery has not therefore taken place when the relapse occurs. Quite suddenly, in the afternoon, in the evening, or more frequently at night, the relapse comes on with a chill which is rather exceptional, or a sense of chilliness, or with fever only. The relapse, as a rule, repeats the symptoms of the initial seizure, except that its course is less severe and of somewhat shorter duration; but the pains, nausea, and vomiting, enlargement of the liver and spleen, are very much the same. The fever has more of a remittent type, and the sweats have a somewhat critical aspect, for more relief is afforded by them than during the primary paroxysm. An attempt at critical phenomena may be made a day or two before the real crisis; there may be a considerable sweat and a marked fall of temperature; but the effect is not maintained and the temperature rises again. The final defervescence occurs from the third to the fifth day, and usually at night, when a profuse sweat occurs, and the temperature and the pulse-rate fall below normal. The crisis may be postponed to the seventh day, but this is not usual. A second, a third, even a fourth relapse has been noted in some epidemics. The symptoms are the same, but the more numerous the relapses, the more reduced must the patient become by a repetition of the suffering.

Course, Duration, and Termination.—The whole course of an ordinary case of relapsing fever is concluded within three weeks, unless there be several relapses. At the conclusion of the relapse, the patient lies in a condition of great comparative comfort, but much emaciated and quite exhausted. The anæmia is very marked, there is more or less œdema of the ankles, the eyelids are puffy, and the sclerotic pearly white. The convalescence is very slow. Much, of course, depends on the violence of the seizures, and the number of relapses. Age appears to have an influence, for, in children under twelve, Parry observed that the course of the disease was shorter and milder. There are also differences in different epidemics in respect to the duration and severity of the disease. The usual termination is in health, the mortality being about two to three per cent. Complications may have a very great influence over the result. Bronchitis, catarrhal pneumonia, and pleuritis, occur in some epidemics, and laryngitis has required tracheotomy. At the period of crisis, hæmorrhages may occur, notably epistaxis and local paralyses—of the deltoid, for example—have been observed. Diarrhœa has occurred at the crisis instead of a sweat—in some epidemics increasing the mortality. A pregnant woman ill with relapsing fever is almost certain to abort, and hence this must be regarded as a serious complication. At the period of crisis, fatal syncope has occurred without any apparent reason. The extraordinary revolution which then takes place may impose too great a strain on a weak heart. The persistence of changes in the liver and spleen, after recovery from the fever, must place these affections among the sequelæ. In the same category is a form of ophthalmia which has occurred after certain epidemics.

Treatment.—The remedial management of relapsing fever must necessarily be expectant. We possess no agent to prevent the development of the *spirilla* in the blood, and we do not know how this parasite enters the blood, or whence it comes. The treatment of the fever would seem to require the use of antipyretics, but their utility is very limited, owing to the short duration of the paroxysm.* The best means of relieving the severe pains are the hypodermatic injection of morphine and the wet pack. Opium by the stomach has but little effect, apparently, in this disease. For the nausea, the best remedy, probably, is carbolic acid (half a grain) administered in cherry-laurel water. For the nocturnal pain and wakefulness, a combination of chloral and morphine promises best. The enormous production of *spirilla* during the paroxysms of fever and their disappearance in the intermission are strong arguments in favor of the administration of parasiticides. The use of quinine has been quite fruitless. But a more systematic administration of the sulphites and the disengagement of sulphurous-acid gas in the air of the sick-apartment should be attempted. At the period of crisis, syncope may be prevented by the timely use of alcoholic stimulants. It is especially during the period of intermission that an attempt ought to be made to prevent the new development of the *spirilla* which it is supposed then takes place. Suitable food, iron, and other tonics should be given to improve the quality of the blood; the increased volume of the spleen reduced, and the overproduction of white corpuscles prevented by the administration of quinine and ergotin, and an attempt made to prevent the new growth of the parasite by the free use of the sulphites and other parasiticides.

YELLOW FEVER.

Definition.—*Yellow fever* is an acute, infectious disease, occurring only south of 48° north latitude, in regions having a mean annual temperature of not less than 70° Fahr., endemic on the seacoast, and sporadic elsewhere under an elevation less than twenty-five hundred feet above the sea-level, the germ being introduced and certain localizing conditions favoring its development.

Causes.—Pursuing the plan heretofore followed, the author will not occupy space with controversial questions. The cases (private) seen by the author occurred in the Mississippi Valley, and were encountered at Cincinnati, having come there from infected localities in the South, especially Memphis. It seems necessary to the production of yellow fever that a peculiar germ or morbific principle be introduced from without. For the further development of this germ it is necessary that there be a concurrence of certain telluric and personal conditions. It is needless to discuss here whether the poison ever arises spontaneously in its natural habitat under the necessary conditions. Of the nature, form, and composition of the morbific principle, nothing is as yet known, and the last investigations in regard to it have proved as barren of results as the preceding one. We know that a mean annual temperature of about 72° is necessary to its activity, and that cold —a frost—suffices to destroy it. A fall of temperature short of that necessary to suspend the activity of the poison increases the mortality from it. Yellow fever occurs in maritime cities first, and extends thence to towns and cities having direct communication with them by river or by railroad. Cities and towns, removed, by reason of their situation, from intercourse with infected maritime cities, escape epidemic visitation. The disease does not spread from city to city so rapidly as men move from one to the other. A germ or germs are introduced. Accumulated filth, decomposing animal and vegetable matters, bad or no drainage, crowding, and other hygienic evils, are indispensable to impart the necessary vitality. Lodging thus in a suitable soil, and with the appropriate atmospherical conditions present, the disease-germs grow and infect those in the proper personal state to receive the poison. After a time, from this newly infected locality, germs are transmitted to other localities. The conditions existing on shipboard seem peculiarly favorable to the growth of the poison. Next to the ship, as a nidus for yellow fever, is the large maritime city, situated at the outlet of a great river, subject to annual overflow and filled with all the materials of insalubrity.* To these must be added the atmospherical peculiarities of July, August, and September. When the disease-germs are introduced, and the localizing conditions are favorable, not all persons are attacked. Some present a peculiar susceptibility, others insusceptibility to the action of the poison. Race exercises a remarkable influence, the pure negro possessing a singular immunity against the infection, provided he has not lived outside of the yellow-fever zone and returned to it just before an outbreak. Any considerable admixture of white blood destroys the protection. Whites are more susceptible the farther removed from the yellow-fever zone they have lived previously. Long residence in the infected locality, especially passing through a period of epidemic prevalence of the disease, and still more effectually passing through an attack, procure more or less complete immunity ; but this immunity may be lost and susceptibility restored by any protracted stay outside of the yellow-fever zone. This process of hardening against the reception of yellow fever is called *acclimation.* It is not by personal contact that the disease is communicated—in other words, it is not a contagious* but an infectious disease, and it is not against individuals that quarantine restrictions should be enforced, but against articles of clothing, bedding, or the like, or against all fomites. The condition of the individual opposes or favors the reception of the poison. Besides all those conditions which favor or retard the spread of the poison above mentioned, must be stated the habits of the individual. All excesses in drinking or venery either help the reception of the poison or increase the virulence of its action in the body. All depressing moral emotions, especially fear, act unfavorably.

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* As this work is going through the press, Dr. Riess, of Berlin, reports that he has found the salicylate of soda remarkably effective in reducing the temperature, and, if given in large doses for some days, will lessen the severity, and even prevent the relapse ("Berliner klinische Wochenschrift," No. lii, 1879).

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* See Dr. Woodhull's (Surgeon U. S. A.) account of "The Causes of the Epidemic of Yellow Fever at Savannah, Georgia, in 1876," "The American Journal of the Medical Sciences," July, 1877.

Pathological Anatomy.—Not much wasting of the body is observed, and the *post-mortem* rigidity is usually well marked. The color of the skin is light or dark yellow, a change which appears to be never wanting in genuine cases. The skin is also stained by hæmorrhagic extravasation, ecchymoses, vesicular eruptions, and gangrenous vesications at points where irritating applications had been made. The dura mater is often yellow, the sinuses engorged, the vessels of the pia congested, rarely hæmorrhage in the subarachnoid spaces or bloody serum, the cerebrum not abnormal, the ventricles containing a little serum, very rarely bloody serum, and similar conditions in the spinal canal, there being nowhere in these organs any evidences of inflammation.† On the other hand, inflammation of the spinal arachnoid in the lumbar and sacral regions has been reported, but the constancy of such lesions must be regarded as doubtful. The changes which have been observed in the cœliac and hepatic plexuses, and which consist in an inflammation of the neurilemma, must also be considered as of doubtful significance.‡ More or less congestion of the lungs, chiefly hypostatic, is usual, and the bronchial mucous membrane presents the usual appearance of passive congestion. The sac of the pericardium contains more or less serum, as a rule, and it is rarely bloody. Purpuric spots are occasionally seen on the pericardium, endocardium, and on the surface of the heart itself. The muscular tissue of the heart may be unchanged, but it is very often more or less softened by granular degeneration. Various changes observed in the composition of the blood are described, but thus far nothing peculiar to yellow fever has been discovered. It is true, Dr. Joseph G. Richardson, of Philadelphia, supposed he had found a peculiar bacterium, which he described as *bacterium sanguinis*, in the blood, but other competent observers have been unable to confirm his observations. A rapid crenation of the red-blood corpuscles has been noted by Dr. Schmidt,* of New Orleans, which he regards as a retrogressive change probably not peculiar to yellow fever. No alterations have been observed in the white blood-corpuscles, although there seemed to be some slight increase in their relative proportion. The most characteristic of the morbid alterations of yellow fever are those of the liver and other abdominal organs. In the Lisbon epidemic, in the epidemics of this country, and elsewhere, the liver has always been remarkably altered. Exteriorly, it most usually presents a fawn-yellow, or buff-color, which is pretty uniform throughout the whole organ, although here and there may be patches of a deeper color. Various shades of the above-described tint are observed in some cases and in different epidemics, because the degree to which the alteration has attained differs somewhat; but when the ordinary liver-brown color is present, on minute examination, the liver is found to be altered in the usual way. The change taking place in the liver consists of a fatty infiltration, and a fatty degeneration of the protoplasm of the hepatic cells. In an advanced case, the hepatic cells are smothered in a mass of fat-cells and granules. More or less coloration of the cells about the radicles of the blood-vessels with blood and bile-pigments is to be seen. The stomach-veins are deeply engorged. This engorgement may be general or partial, and if partial the mucous membrane about the cardiac extremity is chiefly affected. Patches of vascularity, punctiform congestion, ecchymoses, and purpuric spots, have been observed in different cases. The epithelium is usually intact. More or less "coffee-ground" matter, or dark, coffee-colored liquid, containing coffee-grounds mixed with it, is found in the stomach. The black vomit consists chiefly of blood and epithelium; the blood-corpuscles are deprived of their hæmoglobin, which is separate; and the rest is made up of white corpuscles, epithelial cells, and *débris*. The spores and fully developed yeast-plants (*Torula cerevisiæ*) are found in the vomited matters, and other fungi quickly develop in them on standing. The mucous membrane of the small intestine presents the same deep congestion as that of the stomach. In more than one third of the cases in the Lisbon epidemic there was present in the intestine extravasated blood in various stages of the alterations produced by the intestinal juices, and which presented an inky blackness, a reddish-brown or a bloody tint. In quantity the extravasation was sufficient to distend the small intestine in some instances, and was generally considerable. The glandular apparatus of the small intestine has been usually represented as intact in

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* This question is most elaborately treated by La Roche ("Yellow Fever," vol. ii), who finds the arguments against contagion stronger than those in favor.

† Lyons, *op. cit.*, Appendix, "Pathological Anatomy of the Yellow Fever of Lisbon," 1857.

‡ The official commissions appointed to investigate the nature of yellow fever have not contributed any new facts, or made any discoveries that help us to a better knowledge of the disease.

* "New York Medical Journal," February, 1879.

all the various epidemics. No characteristic changes take place in the spleen. The kidneys are rarely normal. A considerable hyperæmia of these organs seems to be nearly constantly present. The epithelium of the tubules undergoes granular degeneration, and this takes place both with the straight and convoluted tubes. Fatty degeneration follows in those cases where death has been long enough postponed to give the necessary time. The urine undergoes important alterations. The uric acid and urea diminish and ultimately disappear, and are replaced by leucin and tyrosin, while albumen appears, at first in a mere trace, but increasing in amount. The urine also assumes a deep color from the quantity of blood-pigment and bile-pigment present in it, and is denser and more viscid (Vidaillet[*]). Schmidt calls attention to changes in the supra-renal capsules, but they do not seem to be different from the appearances observed in numerous maladies.

Symptoms.—*First Stage.*—The period of incubation varies within wide limits, if conclusions are drawn from exceptional cases.[†] Usually, from the period of exposure to and reception of the disease-germ, from one to three days will elapse. The disease begins in two modes—one with prodromic symptoms or gradually, and the other very suddenly. Soon after the reception of the poison, in many subjects, there ensue impaired appetite, a feeling of debility, headache, muscular pains, for two or three days, when the disease sets in with a chill, or a feeling of chilliness followed by fever. In other cases there are no prodromal or premonitory symptoms, and the patient is seized apparently while in full health, walking, at work, asleep, with a chill, sometimes a severe rigor, and the fever comes on immediately. Very rarely have been witnessed in recent epidemics those formidable cases in which the patients in apparently full health were stricken as it were with a heavy bar on the back, falling at once into a condition of profound prostration, and dying collapsed in a few hours. These cases were known as *coup de barre*, or stroke of the bar, because of the intense violence of the sudden pain in the back and loins. In every epidemic, however, there are cases characterized by profound blood-poisoning and rapid termination in collapse. These variations will be mentioned presently. Now we are concerned with the ordinary course of the disease. The fever rises rapidly and reaches its maximum on the evening of the first or second day (103°, 104°, 105°). According to the tracings of Faget, as given by Sternberg,[‡] "in sixteen the acme is reached on the first day; in twenty-three during the first two days"—the whole number of observations being twenty-six. The onset of the disease causes great disquiet, and the victims are restless and disheartened. The face appears anxious and flushed; the eyes moist and bright, and the conjunctivæ injected. There are decided headache, throbbing of the temples, general muscular pains, but especially severe and depressing pains in the back and loins, which in their worst form constitute the dreadful *coup de barre.* Early in the disease, and, according to some, before the outbreak, a peculiar odor is perceived, and by many is regarded as distinctive of yellow fever. The odor is rather cadaveric and diffusible, but much that is asserted in regard to it seems to the author very apocryphal. The tongue is heavily coated with a thick, whitish fur, and is red at the tip and edges, the swollen papillæ projecting above the surface. The palate mucous membrane becomes red and œdematous. The stomach is from the first irritable; the epigastrium is tender to the touch; cold drinks are taken with great avidity, excite pain, and are rejected with a good deal of painful retching at first; and the stomach is equally intolerant of all kinds of food. Sometimes there is diarrhœa, but usually the bowels are constipated. The vomited matters at this early stage consist of particles of food, mucus, and bile, and flocculi of brownish or chocolate colored material—the forerunner of the dreaded black vomit. The stools are pasty and grayish, but constipated. The urine lessens in quantity, darkens in color, and distinct traces of albumen are now discovered in it. The pulse is rapid, strong, with high tension in some cases, weak and dicrotic in others, and the pulsations range from 90 to 120. When the temperature reaches its maximum, usually on the second day, it begins to decline by lysis, a remission occurring about the fourth day, terminating the first stage. In the mildest cases the remission occurs on the second day, and it may be postponed to the sixth day or longer. During the period of maximum temperature and the first stage, besides the symptoms already mentioned, there may be considerable restlessness and active delirium, the patient being kept in bed with difficulty, or the delirium may present the appearance of *delirium tremens*—an active, busy, and trembling delirium. At this stage there may begin to appear a jaundiced tint of the skin; the urine may contain bile-pigment, the stools having a clay-color, which is, however, not usual. There may also occur hæmorrhages from the nose, from the gums, and also from the stomach; but it is only in the severe cases that these hæmorrhages occur so early, and hence they are of evil augury.

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[*] "Archives Générales de Médecine," November, 1869.

[†] La Roche, "Yellow Fever," vol. i, p. 511, Philadelphia, 1855.

[‡] "On the Nature and Duration of Yellow Fever, as shown by Graphic Temperature Charts of Typical Cases, etc.," "The American Journal of the Medical Sciences," July, 1875, p. 99. By Dr. George M. Sternberg, U. S. Army.

Second Stage.—The decline of temperature which marks the end of the first stage may proceed to a complete intermission. In all of the cases collected by Sternberg, "a complete intermission, or nearly so, was found on the morning of the third day." According to others, Haenish for example, there is not a complete defervescence—only a remission—in a majority of the cases. With the decline in temperature there occurs a most favorable change in the condition of the patient. The delirium subsides, the pains cease, the stomach may become quiet, some critical evacuation, as a sweat, an attack of diarrhœa, or an epistaxis, may occur, and convalescence be at once established. Instead, however, of these favorable symptoms the delirium may persist, the irritability of the stomach may increase, albumen, if it has not been in the urine, may now appear, the pulse may become weak, and the condition of the patient may grow rapidly worse, notwithstanding the marked defervescence and the relief to the symptoms which may at first be caused by the remission. The period of time occupied by the remission varies considerably, and is from one to four days.

Third Stage.—The remission disappears and the temperature rises again, but not so rapidly as during the first stage, the maximum of about 104° being reached on the second day. If the active delirium persists, the patient becomes unmanageable, refuses food and drink, the leg-muscles are thrown into violent cramps, jaundice deepens, black vomit comes on, the pulse fails at the wrist, and death closes the scene suddenly in the midst of violent delirium. In much the largest proportion of cases, the mind is unclouded, and the moral state that of complete apathy and indifference. The strength rapidly declines, and the pulse is small, weak, and irregular. The jaundice passes from the characteristic lemon-color to a deep mahogany, and hæmorrhages pour out from the various mucous surfaces and from the skin; the nose bleeds, and blood is vomited, passed by stool, and less often expectorated. The gums are soft, spongy, and bleed with a touch, and rarely the ears bleed. The most striking and characteristic phenomenon is the hæmorrhage into the stomach and the return of the blood in the form of "black vomit." Even during the first stage, small flocculi, of a chocolate-color and composed of altered blood, are seen in the vomited matters, but the "coffee-grounds" do not appear usually until the second, or stage of remission, and often indeed not until the third stage. The urine constantly lessens in amount; the urea disappears; blood-pigment distills through in large quantity; the albumen increases, and very soon, in some cases, entire suppression occurs. Under these circumstances, somnolence, stupor, and ultimately coma supervene. Partial convulsions, hiccough, and Cheyne-Stokes breathing are often observed in these uræmic cases. The temperature also greatly declines toward the end—to 100° even; and it is a curious fact that the action of the heart continues for a time after the respiration and pulse at the wrist have ceased. If the case take a favorable turn during the third stage, the temperature descends to normal very abruptly, and an improvement in the condition of the patient at once occurs. The vomiting stops, and a little aliment may be taken; the kidneys act freely, the circulation improves, and very gradually convalescence is established.

Course, Duration, and Termination.—There are several forms of yellow fever, which differ sufficiently to require some special consideration. Many divisions have been made, but in the following forms are comprehended the most important varieties—the algid, the sthenic, the hæmorrhagic, the purpuric, the typhous (Lyons). The purpuric form is, however, only the hæmorrhagic modified. Excluding this, we have four varieties of the disease, capable of ready clinical distinction. The *algid form* occurs in subjects debilitated by want and misery. The surface is cold, the face sunken and of a livid hue, the extremities blue, cold, and shrunken, the skin covered with purpuric patches, the pulse small and feeble, the temperature in the axilla at 96° Fahr. Such symptoms are not present merely at the outset, but continue to the end. Black vomit occurs early, and the hæmorrhages take place from all the mucous surfaces. In the *sthenic form*, the opposite conditions prevail. The robust, at the prime of life, are the subjects. High fever, severe headache and lumbar pain, delirium of an active kind, early jaundice, having the lemon-tint, and less of the black vomit, are the most characteristic features of this form. In the *hæmorrhagic form*, the peculiarity consists in profuse and simultaneous discharges of blood, effused at various points. Black vomit and intestinal hæmorrhage, uterine and renal hæmorrhage, simultaneous bleeding from the eyes, nose, ears, and mouth, and effusion of blood from any accidental abrasion, give to this form a distinct individuality. In the *typhous form* are presented symptoms which ally these cases to other typhous processes. They are characterized by stupor, prostration, sunken countenance, suffused eyes, dorsal decubitus, low-muttering delirium, in addition to the usual and ordinary symptoms of the disease. The mortality from yellow fever is largely influenced by the type of the prevailing epidemic, and also by the local conditions, and by the form of the seizure, whether algid, hæmorrhagic, sthenic, or typhous. It necessarily varies much, and between such wide limits as from fifteen to eighty per cent. More men die than women and children. The habits of the individual as to temperance enter seriously into the prognosis, since the mortality among spirit-drinkers is very high. All circumstances which act to depress the vital forces increase the severity of an attack. The early occurrence of black vomit and suppression of urine are very ominous symptoms.

Diagnosis.—The only disease with which yellow fever is likely to be confounded is remittent fever with jaundice. The distinction rests on the temperature line and the occurrence of black vomit. The remissions of malarial fever are quotidian or tertian, and the fever of the first stage of yellow fever is continued until the defervescence. Nothing like black vomit occurs in malarial fever; while remittent fever is promptly broken up by efficient doses of quinine, this remedy has no influence on yellow fever. Again, remittent fever prevails much more widely than yellow fever. It is only within the yellow-fever zone that a question of differentiation can arise. When an epidemic influence is at work, there can be no difficulty in the diagnosis after the first cases have appeared.

Treatment.—It is good practice to begin the treatment by a mercurial purgative; a half-grain of calomel two or three times on the first day, followed by a warm-water enema. All drastic cathartics should be avoided, owing to the irritable state of the stomach. If the pain in the back and loins is very severe, one twelfth of a grain of morphine should be administered hypodermatically, and repeated according to circumstances. For the irritable stomach, there are two most efficient remedies, carbolic acid, and lime-water with milk—a fourth of a grain of carbolic acid in some mint-water every two hours, and a tablespoonful of lime-water and milk, equal parts, every two hours, so that these remedies will be taken in alternation every hour. Ice should be kept in the mouth and small pieces swallowed, but care is necessary to avoid distention of the stomach. For the epigastric tenderness, mustard should be applied, and, if the patient is vigorous and the reaction sthenic, leeches or cups should be used. During the second stage, for the irritable stomach a little dry champagne is often very serviceable, as it is very grateful. Hydrocyanic acid, and especially chlorodyne, may also act well as sedatives to the stomach. If the fever is high, the skin hot and mordicant, the wet pack may be used with advantage, or the body may be sponged over and then rubbed with some animal fat, as lard or suet, several times a day. The temperature may be reduced further by the rectal injection of a scruple of quinine, but this agent should not be administered by the stomach, as it will surely excite vomiting. For the same reason all harsh and drastic or irritating medicines should be avoided. The delirium and obstinate wakefulness of some cases require morphine and atropine (the latter in small proportion) hypodermatically. When the delirium is active, the patient restless and difficult to control, the most efficient hypnotic and calmative is duboisine given subcutaneously ($\frac{1}{80}$ grain); Aitken suggests chlorodyne. As digestion is almost entirely suspended, it is useless to push beef-tea and milk when the stomach rejects everything. The best aliment is milk and lime-water, half and half, given in small quantity, not to exceed a tablespoonful every two hours. If curds are thrown up in hard masses, the quantity of milk is too great. Thin barley-water to which some milk is added, and then diluted with lime-water, is a suitable aliment. During the stage of convalescence, the utmost circumspection is necessary in giving aliments. The algid form of yellow fever requires stimulants from the beginning. In the hæmorrhagic form, small doses of turpentine and tincture of the chloride of iron should be given frequently. In the sthenic form, the wet pack, leeches, quinine, morphine, and duboisine, are the most appropriate remedies. In the typhous form, suitable aliment, wine, and the stronger stimulants are required. Yellow-fever patients should be isolated. All of the dejections by vomit or stool should be at once disinfected. The room and halls should be fumigated with sulphurous acid. All articles of clothing and bedding about the patient should be destroyed, or put into boiling water and boiled before handling. Questions of quarantine are not included in the scope of this work.

DENGUE.

Definition.—*Dengue*[*] is an acute febrile disease which prevails as an epidemic, and is characterized by two paroxysms of fever, with an intermission of variable duration between them, the first paroxysm being accompanied by high fever and joint swellings, and an eruption, the second subsiding suddenly with some critical evacuation. It is also called "break-bone fever," "dandy fever," "neuralgic fever," etc.

Causes.—There are close analogies between dengue and relapsing fever; indeed dengue is a relapsing fever. It occurs as an epidemic, and attacks a large part of the population among whom it appears. Apparently beginning on this continent, or in the West India Islands, it has spread to most of the warm countries of the globe, following the routes of human intercourse. Rush, one of the first to give an account of it, mentions it as occurring in Philadelphia in 1780. It is not generally regarded as contagious, although maintained to be by Dickson,[†] and some others. A peculiar condition of the atmosphere seems necessary, the epidemics occurring after prolonged high temperature (Dickson), or great heat and moisture combined (Aitken[‡]). It has been observed in several epidemics that the attacks of dengue succeeded to epidemics of scarlet fever, of yellow fever, and of whooping-cough. The disease occurs in all ages and in both sexes, but the negro race seems to be, although not exempt, somewhat less susceptible, while the mulattoes are attacked equally with whites.

Symptoms, Course, Duration, and Termination.—There may or may not be a prodromal or preliminary stage. The period of incubation is in some instances "prodigiously brief" (Dickson), the attacks in any given household occurring so nearly simultaneously that all are sick at the same time. Toward the end of an epidemic the period which elapses after exposure may be lengthened to five, even to ten days. When the epidemic is at the maximum, the attack may follow exposure within a few hours and the disease occur promptly without any preliminary symptoms. When prodromes occur they consist of weariness, lassitude, headache, anorexia, a white tongue, and more or less general soreness of the body. Usually, however, the onset of the disease is sudden. The patient is taken in full health, often waked out of sleep, with intense headache, burning pain in the temples, backache, and severe aching of all the joints, including the fingers and toes. Sometimes the initial symptom is an acute pain in the knees, ankles, and wrists, the patient being seized while walking. General muscular stiffness follows, so that the affected members become useless, and any attempt to move the joints causes severe suffering. With the headache there is also great intolerance of light and sound. The face is flushed and hot; the tongue coated; a good deal of burning pain is felt in the abdomen; there are nausea and vomiting, during which a quantity of bilious matter comes up, and scarcely anything is retained; constipation persists; the action of the heart is rapid, the pulse strong, and beating at 140 or higher in children. Sometimes also, especially in children, there is delirium, and, in very young children, the onset of the disease may be marked by convulsions (Dickson). An exanthem of very variable character, but most frequently scarlatiniform, may appear, and hence the frequent confounding by the older authors of this disease with scarlatinal rheumatism. The duration of the first febrile stage is very variable, lasting from six hours to several days. It may cease rather suddenly with critical phenomena, or slowly by lysis. The decline of the fever is signalized by the disappearance of the eruption if it had existed, by the appearance of moisture on the skin, a profuse urinary discharge, an attack of diarrhœa, the stools being dark and offensive (Aitken), and by the subsidence of the headache and joint-pains. Usually, at the termination of the remission, the patient is in a condition of very considerable prostration, and, although much relieved, is unable to leave his bed. In other cases, the relief is so great and the strength so well preserved that the patient insists on getting up. The remission may not occur at all. In those cases the joint affection appears with, and the eruptions after, the first symptoms; the fever is continuous, and lasts from five to ten days, when it disappears with critical phenomena. It is by no means improbable that a distinct remission of short duration occurs, but escaped the observation of practitioners unprovided with the means of accurate investigation. The duration of the remission or intermission is not constant, and varies from a few hours to two or four days. During the period of remission there are more or less headache and soreness, and stiffness of the joints and muscles, notwithstanding a very great diminution in the severity of these symptoms; hence it may be concluded that the condition of the interval is rather that of remission than intermission. At the conclusion of this interval, whether of several hours' or two or four days' duration, the symptoms begin again: the fever rises, although not so high as during the first stage; the headache, some muscular soreness, but only occasionally the swollen, red, and painful joints, are felt again; the tongue becomes coated anew, the appetite ceases, and more or less nausea, very rarely vomiting, is experienced. The distinctive peculiarity of the second period, however, is the occurrence of an exanthem—erythematous, roseola-like, rubeolous, lichenoid, etc. Usually, beginning as an efflorescence on the palms of the hand and soles of the feet, it spreads thence over the body. It is often accompanied by intense itching. The eruption may be distinctly localized to particular parts of the body. The duration of the eruption is variable, lasting from several hours to two or three days, and terminates by desquamation of the furfuraceous kind. The subsidence of the second stage is gradual, and the patients are left in a feeble state, requiring months for complete restoration. There may occur other relapses. The joints continue stiff and sore for a long time. It is not surprising that persons attacked with dengue should be much reduced. The fever, severe pains, loss of sleep, inability to take food, the critical evacuations, and the relapses, are sufficient to tax severely the vital resources of the most robust patient. It is never fatal in adults, and it is rarely that children die in convulsions. It is a disease without complications, and leaves behind no sequelæ. The whole duration of fully developed cases is about eight days, of which the first stage occupies three days, the intermission two days, and the last stage three days or nearly so, but the period of convalescence may be protracted over several weeks, because of the weakness, emaciation, and lingering joint swelling and pain, and relapses may several times take place, still further retarding recovery.

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[*] The word *dengue* is pronounced *dangay*.

[†] Fenner's "Southern Medical Reports," vol. ii, p. 384, "A History of the Epidemic Dengue as it prevailed in Charleston in the Summer of 1850."

[‡] Reynolds's "System," vol. i, p. 98, American edition.

Treatment.—As dengue is a specific disease for which we have no specific remedy, it must be treated symptomatically, or in accordance with empirical observation. During several epidemics the use of emetics, carried to the point of free bilious evacuations, was followed by decided amelioration of all the symptoms. Next to the emetic in importance is an efficient but mild laxative. The substitution of more healthy evacuations for the greenish, tarry, offensive stools has also had a good effect on the progress of the disease. Anodynes are needed to relieve the severe pains. It is probable that salicylic acid will have a decided influence over the rheumatic symptoms, which are such prominent features of the malady. If salicylic acid or the salicylates fail, antipyretic doses of quinine should be tried. It is important to maintain free action of the organs of excretion; hence, if the pain is so severe as to demand the administration of morphine, the bowels should be kept open and the kidneys active. As the first stage terminates with some critical evacuation, often with a sweat, the behavior of nature may possibly be imitated and the paroxysms shortened by the administration of pilocarpine. The intolerable itching, so often present, may be allayed by sponging over the part a one per cent. solution of carbolic acid. The joint-pains and soreness of the muscles remaining during convalescence may be removed by the application of galvanism. Tincture of chloride of iron is the most useful chalybeate to be given in convalescence. To restore appetite and digestion, tincture of nux vomica will be found efficient, or a combination of dilute phosphoric acid, pyrophosphate of iron, and strychnine may be administered.

HEAT-STROKE AND HEAT-FEVER.

Definition.—Under the terms *insolation*, *coup-de-soleil*, *sunstroke*, and other synonyms, are included three distinct morbid states: acute meningitis, which is comparatively rare; exhaustion from heat, which is common; and true sunstroke, or *thermic fever*,* or *heat-fever*.

Causes.—As the name implies, this disease is due to the influence of excessive heat, certain other conditions being concurrent. It is not necessary, as is popularly understood, to expose the head to the direct rays of the sun. Artificial heat, and the diffused atmospheric heat, will, under appropriate conditions, develop the disease, yet the direct solar rays are more powerfully causative. The habitual consumption of spirits, beer, and alcoholic beverages, unquestionably predisposes to attacks. Excessive fatigue, overcrowding, heavy and close-fitting garments, are also influential factors.

Pathological Anatomy.—The action of the cause is so sudden, and the disease is so rapid in its course, that time is not afforded for the development of structural changes; nevertheless, there are characteristic appearances. The veins and sinuses of the brain are gorged with blood; much venous stasis exists in the lungs; the right cavities of the heart are distended, and the left are contracted and empty. The condition of the heart is disputed. It is generally said to be flaccid, but, according to Wood,† it is firmly contracted immediately after dying, but becomes relaxed as putrefactive changes come on. The blood is fluid, dark, sometimes grumous, feebly alkaline, neutral and even acid in reaction. The red corpuscles appear to undergo in some cases the change known as *crenation*.‡ *Post-mortem* rigidity sets in at once and is very great.

Symptoms.—The acute meningitis caused by heat is the same as that due to other causes, and, as it has been described elsewhere, need not be taken up again. The condition of exhaustion produced by heat has been observed on a large scale by the author, § and, as it is often confounded with the true *coup-de-soleil*, should receive some consideration. During the course of some exhausting labor under a hot sun, as marching equipped with blankets and accoutrements, there comes on an increasing sense of weakness; presently the sight grows dim, a rushing noise sounds in the ears, and the individual falls suddenly or sinks down, partially or entirely unconscious. In the most severe cases the man falls suddenly, or stumbles once and then falls unconscious; a shudder or tremor passes over the body, and sometimes a general convulsion occurs as in the syncope from loss of blood. The face is pale, the features are sunken, the pupil is dilated, the surface of the body is cool and perspiring, the muscles are relaxed, the pulse is feeble and quick, and the respirations are hurried and shallow. The senses are obtunded, the perceptions dull and confused, or the consciousness is wholly lost. Under rest and appropriate treatment the symptoms subside in a few hours, and recovery is effected in twenty-four hours.

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* "Thermic Fever, or Sunstroke," by H. C. Wood, Jr., M. D. Philadelphia: J. B. Lippincott & Co., 1872. Boylston Prize Essay, p. 34.

† Ibid.

‡ Dr. Levick, "Pennsylvania Hospital Reports," 1868, p. 373.

§ The author in 1857 accompanied an infantry regiment, about eight hundred men, on the march to Utah from Fort Leavenworth on the Missouri River. The command started on the 18th of July; the heat was great, and large numbers of men fell out exhausted on the first few days. The drinking, dissipated men were the victims almost exclusively. There were no deaths from this cause.

The attacks of sunstroke are often preceded by prodromic symptoms. The patient experiences frontal tension, headache, and vertigo, and is weak; a strong sense of oppression is felt at the præcordia, and there may be nausea and vomiting. When the attack occurs, the patient may pass suddenly or more slowly into a condition of unconsciousness. Notwithstanding the existence of headache, vertigo, and nausea, the laboring man continues at work, and suddenly falls and quickly becomes unconscious. A soldier on the march, or standing at "attention," may undergo the same experience. Another man, feeling the same prodromal symptoms, may be in a position to lie down, and hence, when apparently asleep, passes into unconsciousness. In still other cases the condition of unconsciousness is preceded by delirious acts; the patient tries to escape from, or takes up arms against, an imaginary enemy; or the onset is announced by a peal of wild laughter. Very often there is present an intense desire to micturate. In some fulminant cases, when insensibility occurs, the patient gasps a few times, a shudder runs over the body, and the heart stops. These have been called the cardiac variety of sunstroke (Morehead). In such cases, owing to the sudden death by syncope, there is no time for the development of the symptoms pertaining to the period of unconsciousness. Usually, these symptoms are present, and are about as follows: The face is flushed, deeply suffused, or cyanosed, and the whole surface presents the same appearance; the conjunctivæ are injected; the pupils most frequently contracted, but may be dilated or normal; the breathing is rapid, noisy, and shallow, or it is labored and stertorous; the pulse is very quick and bounding, or it is feeble and quick; the skin is dry and hot, really mordicant, and the temperature of the axilla ranges from 105° to 110° Fahr., most frequently at 108° or 109°. In most cases, there is a condition of complete muscular resolution, and no movements of any kind take place, all of the reflexes being abolished. In a smaller number, *subsultus tendinum* is present, or restlessness and agitation, or there are clonic convulsions with tetanic rigidity, or epileptiform seizures. In a few cases, petechiæ appear, or hæmorrhages from the mucous surfaces. Involuntary discharge of fæces is the rule.

Course, Duration, and Termination.—The cases of exhaustion from heat usually terminate in health after twenty-four hours, under repose and proper treatment. The prodromal stage of heat-fever lasts a few hours. The fulminant form may end fatally within a few minutes. The ordinary form continues from half an hour to five or six hours. The mode of dying in the fulminant cases is at the heart; of the ordinary form, at the lungs. Those attended with convulsions usually die at the head. In the cases going on to a fatal termination the temperature rises, the action of the heart becomes more rapid and feeble, the conjunctivæ are more injected, the cyanosis deepens, the extremities grow more livid, and the muscular resolution increases in depth. When recovery is about to occur, the surface becomes cooler and is less cyanosed; the respiration deepens, the pulse declines in number and gains in volume, the reflexes are restored, restlessness replaces relaxation, and the convulsive phenomena disappear, if they have occurred. The mortality varies under the varying conditions of the attacks. It is greatest in the old, and in those with damaged heart, and in the obese. The mortality rates as given are vitiated by confounding heat-exhaustion with heat-fever. The mortality may be stated at from ten to fifty per cent.; the latter, however, is nearer the truth in respect to heat-fever. The cause of death being the disorganization of the blood produced by the hyperpyrexia, the failure may occur at the respiratory center, at the cardiac ganglia, or at the lungs.

Diagnosis.—The most important question is the distinction between heat-exhaustion and heat-fever, for on this rest the indications for treatment, and success or failure will depend on the direction taken by our remedial measures. In heat-exhaustion, the surface is pale, cool, and relaxed, the temperature being rather below than above normal, and the insensibility is due to syncope and cerebral anæmia. In heat-fever the surface is suffused or cyanosed, the skin hot, the temperature rising into hyperpyrexia, and the insensibility is due to disorganization of the blood. Heat-fever is to be diagnosticated from the insensibility due to acute alcoholism, to opium narcosis, and to cerebral hæmorrhage. The difficulty is great in differentiating between heat-fever and acute alcoholism, because so many alcoholics fall prey to sunstroke. The history is important. The man with sunstroke has been laboring or walking in the sun or heat, when attacked, and he has had the usual prodromes of the seizure. The thermometer must be invoked to decide; in the case of alcoholism, the temperature is rather below than above normal. In the further progress of the cases they are differentiated by the gradual recovery from alcoholic insensibility and by the much more speedy termination of the case of heat-fever. The distinction from opium narcosis rests on similar grounds. The minutely contracted pupil, the slow respiration, the cold surface, are the opposite of the rather dilated pupil, noisy and rapid respiration, and high fever of heat-stroke. In the insensibility of cerebral hæmorrhage, the conjugate deviations of the eyes, the slow, full pulse, the labored respiration, the low temperature of the surface, and the preservation of the reflexes in many cases, serve to distinguish this state from heat-fever.

Treatment.—Heat-exhaustion requires rest and stimulants. The head should be low, and the body recumbent. If the patient is able to swallow, he should at once receive an ounce or two of brandy and thirty minims of tincture of opium; or, if unable to swallow, these remedies can be thrown into the rectum, or some whisky and ten to fifteen drops of tincture of digitalis can be injected subcutaneously. It need not be remarked, probably, that bleeding and the application of ice are entirely inadmissible.

Different methods are required in the treatment of heat-fever. The extraordinary temperature, on the persistence of which the danger depends, must be speedily reduced. Rubbing the body with ice, the cold bath, the wet pack, or the cold douche, are the means most effective for this purpose. In India the practice consists in removing the patient to the shade, and at once douching the whole body, stripped, with cold water. This speedily reduces the temperature. The tendency to subsequent elevation of temperature is best obviated by wrapping the patient in the wet sheet. Cold water may also be thrown into the rectum. If depression come on, some whisky or brandy may be given. The subcutaneous injection of quinine may also be practiced to reduce the heat. In cases characterized by restlessness and convulsive phenomena, morphine, hypodermatically, has been used with great success.* Inhalations of chloroform have been administered with equal success under the same circumstances. But the inhalation of chloroform and the hypodermatic injection of morphine may be pushed too far. On the whole, the injection of morphine is the safer expedient, and, the facts show, is very successful in suitable cases. One fourth of a grain of morphine sulphate is a proper dose for a robust adult. As extraordinary tolerance of the remedy is often exhibited under these circumstances, an amount of it may be given which would prove fatal in health. If the convulsions subside, the breathing become more tranquil, and the temperature decline under the use of the injection, it is doing good, and may be repeated, if necessary, in a few minutes, but it is better to await the action of the half-grain until it is clear that a third dose will be necessary. The administration of morphine subcutaneously is not incompatible with the use of the cold douche, wet sheet, and other measures required to abate the high temperature. The occurrence of sudden depression of the powers of life, the patient passing into collapse, is an unfortunate tendency in some of the cases, which may be attributed to the treatment used. The practitioner should be on his guard, not only to obviate this tendency by the timely use of stimulants, but to avoid reproach.

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* Dr. Hutchinson, "Pennsylvania Hospital Reports," *op. cit.*

MIASMATIC DISEASES.

CHOLERA.

Definition.—*Cholera* is an acute infectious disease, endemic in some localities, epidemic elsewhere, and characterized by vomiting and purging of a peculiar rice-water-like fluid, and a condition of collapse and death, or of a reaction from collapse and the development of a typhoid state. It is known also as *epidemic cholera*, *Asiatic cholera*, *malignant cholera*, etc.

Causes.—The etiological factors concerned in the diffusion of cholera are very complex. Is there a cholera-germ? The facts thus far accumulated render it highly probable that cholera is propagated by a minute organism—according to Koch, a *bacillus*, the *comma bacillus*. Although some eminent bacteriologists refuse to accept this view, the weight of testimony is in its favor, and as the crucial test of cultivation, and the production of a cognate disease in animals, is not wanting, we can hardly refuse our assent, until, at least, another organism is finally proved to be the morbific agent. When the first epidemics of cholera started on their march around the world, they pursued a general direction from east to west, following the routes of commerce, and from one great center of population to another; but this course was not inevitable from the nature of the poison, and it is now known that the disease pursues no defined course, and, in fact, spreads in all directions, according to the freedom of communication. It is conveyed by caravans, by ships, in clothing, baggage, and other effects, by streams of water, by air, etc. It is not contagious, in the common acceptation of that term. Physicians and attendants in cholera hospitals are not more exposed than others, during the existence of the epidemic, unless a local source of infection occurs. The author had charge of the cholera hospital in Cincinnati during the epidemic of 1866, and not only visited the wards several times daily, but made a number of autopsies, and on several occasions was wounded, without experiencing the first symptom of the disease. The assistant physicians and attendants were equally exempt. The dead bodies of cholera subjects apparently possess no infective property. The bacteria of decomposition destroy the disease-germs of cholera. The morbific material or germ is more certainly conveyed in the moist state, and some preparation or transformation must be undergone before it becomes active. As it leaves the person of the sick it does not appear to have toxic power, but acquires this subsequently. Hence cholera is not communicated directly from one person to another: an intermediate condition of preparation is necessary. Hence the importance of the superficial water-supply (the *ground-water* of Pettenkofer), and of certain geological formations. The character of the soil best adapted to the nurture of cholera-germs, because retentive of the surface-water, is alluvium, light and porous, resting on an impervious clay subsoil. Malarial regions are generally very favorable to the growth of cholera-germs. When the ground-water is low, the germs are produced in greater abundance than when it is high. Cholera is always spread rapidly when the drinking-water is supplied from the surface drainage, and hence is rich in organic matter. The records of cholera epidemics are full of most striking examples of this truth. The excretions of cholera patients, thrown on the ground, or into superficial privy-vaults, quickly reach the ground-water, multiply rapidly, and soon the sources of water-supply, the superficial wells and streams, become contaminated. Hence it is that one of the principal sources of cholera infection is the water-supply. When an epidemic influence prevails, not all exposed to the poison contract the disease; great differences in the individual susceptibility are found to exist. The hygienic influences affecting the individual are highly important. Excesses in venery, in spirit-drinking, late hours, and an irregular life generally, bad air, and moral depression and fear of the disease, exercise an unfavorable influence. Males are more apt to have cholera than females, and infants are less susceptible. The mortality is less among children than among adults, and is greatest between twenty and thirty. Although it is true that heat favors the spread of cholera, and that the greatest mortality is during the hot season, yet it does prevail during the winter; a notable example was afforded by the Russian epidemic of the winter of 1830–'31. The disposition to an attack of cholera seems greatest in the early morning. A hot, moist, and stagnant atmosphere is especially favorable to the development of the epidemic influence. A light rainfall, followed by a warm mist, the air being still, was the condition of the atmosphere when the cholera assumed its most severe phase in the Cincinnati epidemic.* An ordinary epidemic, under the circumstances of its introduction in one of our cities, is not likely to prevail longer than two months. July, August, and September are the months of greatest prevalence of the epidemic, as a rule. From the period of exposure and reception of the poison until the outbreak of the disease —the incubation—from two to four days usually elapse. But this is not a fixed and invariable period—it may extend to one or two weeks, but very rarely longer. Healthy persons, arriving in an infected city, are attacked in from three to four days. When the germs of disease are brought to a healthy city, about a week elapses before cases of the disease appear.

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* A "norther," with rain, preceded a fearful outbreak of cholera among the United States troops (Eighth Infantry) at Lavacca, Texas. Reported by Dr. N. S. Jarvis, U. S. A., Fenner's "Southern Hospital Reports," vol. i, p. 436, *et seq.*, 1849.

Pathological Anatomy.—If death has occurred in the asphyxia, the stomach contains more or less of the whey-like material of the cholera-discharges—a material alkaline in reaction, albuminous, and full of cast-off epithelium. Later, or during reactionary fever, the mucous membrane is congested, and marked by extravasations and ecchymoses. The small intestines usually contain a large quantity of the whey-like fluid, full of epithelium. The glands of Brunner, the solitary and agminated patches are thickened and very prominent. The villi of the mucous membrane, as well as the epithelium, are stripped off, leaving the basement membrane for the most part bare. The solitary glands of the large intestine are also infiltrated and swollen. Sometimes the colon is the seat of a diphtheritic process, but this is a change pertaining to the fever of reaction. The spleen is small, wrinkled, and firm during asphyxia, but in the secondary fever it enlarges and is softer. The biliary passages contain a quantity of cast-off epithelium, which probably obstructs the outflow of bile—for usually the gall-bladder is well distended with a rather thick, viscid bile. The liver is more or less advanced in fatty degeneration, but is not conspicuously altered, although, by reason of changes in the hepatic cells in spots, the organ may present a somewhat mottled, yellowish discoloration, mixed with brown. The changes in the kidneys are of the same nature as those of other mucous surfaces. The epithelium of the tubules is granular, cloudy, and is detached from the basement membrane, blocking the tubes, so that the whole organ has the appearance of the pale, smooth, white kidney. Here and there, however, there are spots of injection, and occasional patches of ecchymosis. The bladder is empty and contracted, or contains a very little milky urine. The peritoneum is dry, sticky, from the presence of a quantity of loose epithelium still adherent, and hence the membrane does not present the transparent and glistening appearance of health. The pleura presents the same conditions: its transparency is impaired, it is adhesive, and the epithelium is cast off in great quantity. The lungs are deeply congested, especially posteriorly; ecchymoses of the bronchial mucous membrane and infarctions of the lungs are occasionally encountered. The great venous trunks and the right cavities of the heart are distended with blood, while the left cavities are empty and contracted. The blood is dark, almost black in color, thick and viscid, feebly coagulable, and sometimes incoagulable. The pericardium is dry, and there are numerous ecchymoses on the visceral layer. The muscular tissue of the heart is not affected. There are but few changes in the brain. The author observed, in all of his autopsies, considerable hyperæmia and dilatation of the vessels of the medulla oblongata. The constancy of this lesion would seem to indicate a relationship between congestion of the medulla and the cramp.

Symptoms.—*First or Prodromal Stage.*—As there are two forms of disease from which cholera may proceed, although they are quite independent affections under other circumstances, they may be with propriety regarded as modes of manifestation of cholera-poisoning. These maladies are *diarrhœa* and *cholerine.* During every epidemic of cholera, a large proportion of cases set in by a diarrhœa, which if permitted to continue will develop into a typical attack of cholera. Others begin as a cholerine, with vomiting and purging like an ordinary cholera morbus, and if uncontrolled the case assumes the characteristics of cholera. Cholera-diarrhœa may arise from ordinary causes —from taking cold, errors of diet, etc. There is some chilliness, thirst is exacting, the tongue is pasty, and there is a bitter or mawkish taste. Some pain may be felt in the abdomen, but the stools pass with ease, are copious and watery, and cause a decided feeling of weakness. There may be no more than two or three stools in the course of the day, but the failure of strength is remarkable and quite out of proportion to the loss of material. Such a diarrhœa may in a day or two become very profuse, the stools whey-like, cramps in the legs, cold tongue, cold breath, toneless voice, suppression of urine come on, and the patient pass into cholera asphyxia. During a cholera epidemic there is danger that every case of diarrhœa may assume cholera characteristics. It has usually been observed that during a cholera epidemic there is a general prevalence of diarrhœa, or such a state of relaxation of the bowels that a laxative causes drastic effects. Cholerine behaves as an ordinary attack of cholera morbus, except that the discharges have less and less of the stomachal and fecal characters, that cramps are more apt to occur, and that the symptoms of cholera asphyxia readily come on. In many epidemics prodromes have been observed. The author has seen, in most cases, mental depression, fatigue of body, and chilliness precede the regular attack. On the other hand, a feeling of recklessness, or apathy and indifference, has been noticed. In all cases diarrhœa or cholerine has ushered in the attack. The characteristics of the diarrhœa have been copious, watery, rapidly becoming whey-like stools, passed easily, with force, and without pain. A majority of patients are attacked after midnight and toward morning. If there had been no diarrhœa the day before, which is rather exceptional, the patient is waked with an urgent desire to go to stool, and he at once passes an ordinary diarrhœa stool of great volume, and the first is quickly followed by others, even more copious and assuming a lighter color. If diarrhœa has existed during the previous day, the first stool is of a whitish color.

Second Stage.—With the large evacuations which announce the onset of the regular cholera attack, there is a marked degree of chilliness, anxiety, and alarm, but with many an absolute indifference. The evacuations come with a rushing force and amount to quarts of grayish, or whitish, rice-water or whey-like fluid. The patient feels cold, weak, and dizzy, and is glad to throw himself on the bed after one or two of these evacuations. It is not long before vomiting sets in, if the attack did not begin as a cholerine. In an hour or so the stomach becomes uneasy and vomiting begins—first, the contents of the stomach and some bilious matter, and then the peculiar rice-water discharges — an alkaline fluid containing flocculi, which subsiding are found to be composed of epithelium, ammoniaco-magnesian phosphate, blood-corpuscles, bacteria, and various minute organisms. Sometimes the quantity of blood-corpuscles present is sufficient to give the whey-like fluid, vomited and purged, a distinctly reddish hue. In every epidemic there are cases sinking rapidly without vomiting or purging, all the other phenomena being present. These are called *cholera sicca*, but incorrectly so, since in the intestines after death are found in great quantity the characteristic discharges. The vomiting is generally less frequent than the purging, and the quantity thrown up less. The vomit is thrown up with force and ejected a great distance. There is intense thirst, and great draughts of water are swallowed, to be quickly returned. The tongue is white, pasty, and cold. The countenance shrinks, has a leaden hue, and the eyes are staring, the nose pinched, and the breath cool. A good deal of præcordial anxiety is felt and breathing is oppressed, even difficult, the respiration sighing, or a troublesome hiccough comes on. Very soon cramps are felt in the calves of the legs, and although they occur in the arms, hands, masseters, muscles of the back and abdomen in many cases, they are more severe in the calves than elsewhere. The temperature rapidly falls. At first the pulse is a little accelerated, but it soon declines in volume and force, becoming extremely small, barely perceptible, or ceases at the wrist, while the action of the heart can hardly be recognized. The surface gets cold and is covered with a sticky perspiration; the skin loses its elasticity and wrinkles, so that the hands have the sodden look known as the "washerwoman's hands"; the fingers, the face, and the nose and lips especially, are blue as well as cold; the eyes are sunken and are surrounded by livid, almost black rings; the tongue is now like ice and the breath is cold; the voice is weak, husky, and sepulchral, and the urine is suppressed entirely, or diminished to a few drops, which is often found to be albuminous. The temperature of the body descends to the level of the surrounding media—to 96°, 92°, even 80° sometimes. The minimum, according to the author's observation, was 92° Fahr. Such is the *algid stage* of cholera, or *cholera asphyxia.* It is a remarkable circumstance that patients reduced to this low point, collapsed and barely living, the blood thick and hardly in motion, should yet preserve their faculties, and, when roused, return correct replies to the queries addressed them. The termination of this state is usually in death, but reaction may be established, introducing the *third stage.*

Death rarely occurs in less than twelve hours from the beginning of symptoms. The state of collapse may last from twelve to forty-eight hours and even then recovery ensue, but, of course, recovery is exceptional under such circumstances. Again, death may occur in three or four hours. When reaction takes place, the pulse returns at the wrist slowly, and at first doubtfully, the surface very gradually becomes warmer, the countenance assumes a more natural appearance and the cheeks acquire a faint flush, the tongue is less cold, there is less thirst, the respirations are deeper and easy, and the temperature rises. The vomiting and purging lessen materially, or cease altogether, but, as vomiting and purging cease in the final collapse, this latter condition should not be mistaken for the former. The secretion of urine and the substitution of normal fæces for the rice-water discharges, above all other symptoms, announce the beginning of convalescence. If albumen be present, as is usual, it gradually diminishes and disappears in three or four days. The return to health may occupy a few days only, but more frequently a week or more will be required. The reaction may not be complete. The stomach continues irritable, thirst is incessant, and indulgence in drinking speedily excites vomiting. The tongue continues coated, or peels off, leaving a dry and glazed surface. The epigastrium remains tender, and the blandest food excites pain and is apt to be rejected. The bowels do not act well. The stools are rather grayish and mixed with bilious-looking matters without having the appearance and odor of fæces. The urinary secretion increases in amount, but there is considerable albumen present. There is also much headache, and, while a condition of somnolence is tolerably constant, there is little genuine sleep, and the mind is clouded with illusions and hallucinations. This imperfect reaction may terminate in recovery, which is by no means frequent, or some acute, intercurrent disease may arise, or the patient may lapse into cholera typhoid. The reaction may pass beyond normal, and convalescence be delayed by fever, by continued irritability of the stomach, and irregularity of the bowels. The eyes are watery, the cheeks flushed, and the face is spotted ; more or less headache, tinnitus aurium, and wakefulness is experienced. After some hours, or a day or two, these symptoms may subside and convalescence be established, or they may pass on into the cholera typhoid. Under this designation of *cholera typhoid* is meant a typhoid state compounded of reactionary fever and uræmia. When health is restored, the albumen disappears in three or four days, but in protracted convalescence the albumen persists, varying in amount from traces to ten per cent. When the state of cholera typhoid is developed, a condition of great debility ensues; there are severe headache, deeply injected conjunctivæ, vertigo, and stupor. They lie in a condition of somnolence, muttering unintelligibly. The tongue is coated, sordes accumulate about the teeth; there are thirst, nausea, sometimes vomiting; the abdomen is distended, and gurgling can be induced by pressure over the ileo-cæcal valve; there is diarrhœa, the stools being greenish and liquid, or constipated, or these states may alternate. Eruptions, sometimes like roseola or like urticaria, or erythematous, appear on the hands, and spread thence over the body. Cramps are apt to occur, and there may be convulsions in children. In the fatal cases, stupor deepens into coma, the pulse fails, the discharges are involuntary, and death occurs in collapse. On the other hand, should recovery take place, the stupor and hebetude of mind clear up, the albumen disappears from the urine, the vomiting ceases, some appetite returns, and digestion is slowly resumed. So damaged have been the organs of digestion, and lowered the composition of the blood, that convalescence is tedious, some weeks being consumed in the work of restoration. Convalescence is often complicated by bed-sores, boils, or carbuncles, by diphtheritic exudation of the fauces or larynx, by bronchitis, pneumonia, parotiditis, etc.

Course, Duration, and Termination.—The course of cholera is quite varied: it includes a period of incubation, a prodromic stage, the first stage, or invasion; the second stage, or algid stage; the third stage, or reaction; and the fourth stage, or convalescence. The period of incubation is irregular, and varies from one day to a week. The prodromic period lasts from a few hours to a day or two. The average duration of fatal cases is about sixty hours, and of cases that recover, about nine days. Death does not often occur within the first twelve hours, but in the algid condition. The usual duration of the typhoid stage is from two to nine days, but the stage of reaction, which precedes the typhoid, may inaugurate speedy convalescence, and terminate by the fifth or sixth day. The mortality from cholera in all countries is singularly uniform, the average of various epidemics being about fifty per cent. In some epidemics the mortality is as high as eighty per cent.; in others, as low as twenty or thirty per cent. The last epidemic in this country was much less formidable, and the disease seemed milder than former ones. In fact, each visitation since the first in 1832 has manifested less virulence than the preceding one. The cholera-germ seems to be naturalized to the Mississippi Valley, for every year since the last great epidemic numerous cases occurred in all respects like those during the spread of epidemics. The mortality is generally greater at the beginning of an epidemic than at its close. Of the large number brought under the cholera influence during an epidemic prevalence of the disease, but few comparatively are attacked. In many the germs received into the intestines excite no disturbance; in others, there is produced merely a cholera-diarrhœa; in still others, a fully developed cholera-seizure follows. The prognosis is influenced by age, habits of life, and hygienic surroundings. Infancy, old age, a debilitated constitution, evil habits, especially alcoholic excess, and living amid the most active sources of infection, greatly increase the danger of an attack. In an attack of cholera the prognosis must rest on the condition of the individual at the time of the seizure, and on the severity of the attack, the prompt development of the algid state being especially of evil import. The signs of evil import during the stage of reaction are imperfect reaction, confusion of mind, suppression of urine, and involuntary discharges. If reaction is well established, and instead of convalescence cholera typhoid comes on, the condition must be regarded as unfavorable, although recovery is not impossible.

Treatment.—It is important to recognize diarrhœa and cholerine as portions of the morbid complexus. No case of diarrhœa is undeserving of attention during the existence of a cholera influence. The great remedy is opium; its importance is testified to by the fact that this agent, in some form, enters into all the cholera remedies, secret and published. As the cholera-discharges are distinctly alkaline, and as inward osmosis can only be properly set up by the administration of an acid, this physical fact should be recognized in the prescriptions. Experience is in accord with theory in respect to the value of an acid. The following combinations for the cholera-diarrhœa the author has found very effective: ℞. Acid. sulphuric. aromat., tinct. opii deodorat., āā ℥ j. M. Sig. Ten to thirty drops in water every hour or two. ℞. Acid. sulphuric. dilut. ℥ ss., tinct. opii camphorat. ℥ jss. M. Sig. A teaspoonful, well diluted, every half-hour to every two hours. Paregoric, fortified by tincture of opium, is an efficient remedy. Many prefer acetate of lead and opium in pill-form, or in solution. A favorite combination is spirits of chloroform, tincture of rhubarb, tincture of cinnamon, and tincture of opium. One of the most successful remedies for the preliminary diarrhœa is the proprietary medicine chlorodyne, which has been largely used in the East Indies. According to Brown-Séquard, who bases his practice on experience acquired in the West India Islands, cholera can certainly be prevented by giving sufficient morphine in time. If the attack begin by cholerine, there is no remedy so efficacious as the hypodermatic injection of morphine and atropine ($\frac{1}{6}$ grain of morphine and $\frac{1}{120}$ grain of atropine). Indeed, it may be affirmed that the subcutaneous injection of morphine is the most efficient treatment of both forms of preliminary disturbance and of the first stage of the attack proper. Besides the medicinal remedies for this stage of the disease, the utmost quiet must be enjoined. The food taken should consist of boiled milk, a soft-boiled egg, some beef or mutton broth, or a moderate quantity of steak or roasted beef. If the symptoms be threatening, the aliment should not include any solids. As thirst is excessive, the patient should be allowed ice *ad libitum*, which he should be encouraged to swallow frequently in small quantities. Effervescent drinks are extremely grateful and very useful when the vomiting begins. Fermented drinks, as beer and champagne, are objectionable, but carbonic-acid water and effervescing powders are, on the other hand, very serviceable. Recognizing the fact of the alkalinity of the discharges, we should give an acid reaction to the effervescing powder by increasing the relative proportion of acid. Mustard to the epigastrium, or a flying-blister will aid in the arrest of vomiting. The subcutaneous injection of morphine is still more efficient. The author must here strongly insist on the futility and danger of deep vesication so often practiced in cholera, for he has seen an inflammation of all the tissues of the abdominal wall, extending to the peritoneum, produced by blisters to the abdomen in the algid stage. Other remedies for the vomiting are carbolic acid, which often acts very admirably, chlorodyne, hydrocyanic acid, tincture of camphor, chloroform, nitrite of amyl, chloral, etc. Of all the remedies for this stage, the author has had the best results from the hypodermatic injection of chloral—of which a scruple may be injected every hour or two, dissolved in a sufficient quantity of water. It allays the cramps, and brings about reaction. It seems to act most efficiently when administered with morphine, or in alternation with the latter remedy. Good effects have followed the injection of atropine in the algid stage, to excite the heart to action, and to restore warmth to the surface. Amyl nitrite has been used by inhalation to obtain the same effect, and apparently with advantage. When the heart is failing and the surface becoming cold, there is a strong temptation to the free use of stimulants, and the stomach is kept full of brandy, camphor, ether, ammonia, and other stimulants. As these articles can not be absorbed, they serve to keep up vomiting. As the circulation declines, a little brandy will be useful, but any considerable quantity should not be given. Whisky can be thrown under the skin. The intravenous injection of milk has proved successful in the hands of Hodder, in the collapse of cholera, and the intravenous administration of salines has, in apparently desperate cases, brought on reaction, but which, unfortunately, is not always maintained. In this direction must be looked for the most successful management of the algid stage of cholera in future epidemics. During reaction the stomach must be handled very cautiously, lest vomiting be excited. The digestive powers are so feeble that it is useless to give any food except a little hot milk or a little weak broth. The vomiting and diarrhœa which are so troublesome at this time are probably best relieved by carbolic acid and bismuth (℞ Acid. carbolic. gr. viij, bismuthi subnitrat. ʒ ij, mucil. acaciæ, aquæ lauro-cerasi, āā ℥ j. M. Sig. A teaspoonful every hour or two). If there are fever and headache, bromide of potassa will give relief. As the cholera typhoid is a condition of uræmia, efforts should be directed to restore the urinary secretion, and the treatment ought to be conducted according to the principles already laid down. As it is probable that the poison of cholera is contained in the discharges, these should be disinfected at once by a strong solution of the chloride of zinc. The linen about a patient, experience has shown, is peculiarly dangerous. When the loss is not important, disinfection by burning should be practiced; otherwise the material should be thrown into boiling water, and should not be handled until thoroughly boiled. Articles of clothing should be hung up in an atmosphere of sulphurous acid for a number of days. During the existence of an epidemic, the hours should be regular and all excesses avoided. The mistake made by changing from a full to a very restricted diet has cost many lives. The ordinary fruits and vegetables of the season should be taken in moderation. Everything indigestible should be avoided. Calmness favors health, while fear invites disease. Attention to the first indications of disease may save an attack. Questions of public hygiene are not embraced within the scope of this work.

DIPHTHERIA.

Definition.—*Diphtheria* is an acute, specific, contagious disease, beginning by an infection of the throat, and characterized by a local exudation, and glandular enlargements, systemic poisoning, and having for its sequelæ various paralyses.

Causes.—As diphtheria is a communicable and an inoculable disease, it is propagated by a specific poison, the form of which is not known, although suspected to exist as a minute organism. The simultaneous discovery by Hueter and Oertel of a minute organism of the bacteria group, in the exudation, the mucous membrane, neighboring vessels and lymphatics, and in the blood, at once attracted attention to this parasite as the infecting principle. Virchow's discovery of the presence of micrococci colonies in ulcerative endocarditis and elsewhere furnishes strong support to this theory of diphtheria. On the other hand, the filtration experiments of Burdon-Sanderson have cast serious doubts on the immediate agency of micrococci; they seem rather to enact a secondary *rôle*, but, according to either position, they are necessary to the diphtheritic process. Diphtheria prevails as an epidemic; under some circumstances it is endemic, and it also occurs sporadically.

Diphtheria is closely allied to scarlet fever, and it occurs during the course of measles, small-pox, typhus, puerperal fever, exudations developing in the fauces during the progress of these diseases, and on the genitalia in the last mentioned.* Indeed, it seems well established that the materies morbi of these low forms of fever favor the development of the diphtheria-poison. While the disease occurs more or less throughout the whole range of civilization, it is more prevalent in the temperate regions. It is more apt to prevail as an epidemic during the winter and spring, but epidemics have occurred at all seasons. Like all other diseases of the same kind, all the conditions of bad hygiene increase its virulence and favor its diffusion. Unquestionably, the chief cause of its spread is contagion. Many nurses and physicians have fallen victims to their devotion. "When it breaks out in a family, all the children are commonly affected with it, if the healthy are not kept apart from the sick ; and such adults as are frequently with them, and receive their breath near at hand, seldom escape some degree of the same disease." † The experience of the last century is the same to-day. As a rule, the more severe the case of diphtheria, the more intense the activity of the poison. When there are several bad cases in a small apartment not ventilated, the poison becomes denser and more virulent, and conversely, when there is a single case in a large, well-ventilated apartment, the poison is diluted, and its virulence lessened. The young, above one year, are more susceptible than adults, the greatest mortality being attained from the second to the fifth year. Boys seem more apt to get the disease than girls, a fact which Fothergill noted in the epidemics of the middle of the last century. An acute catarrh of the fauces seems to invite the contagion, and although one attack does not confer an immunity against subsequent attacks, a considerable interval occurs between them. When we hear of children having diphtheria every year, we have a right to assume that errors of diagnosis have been committed. The poison of diphtheria exists in the exudations and secretions of the fauces, and it is chiefly by means of this that the disease is communicated. Those engaged in swabbing the throat receive this matter as it is ejected in coughing, or with the exhaled breath. Several physicians have been poisoned by blowing through a trachea canula. Articles of clothing may contain particles of matter for a long time adherent to them. Doubtless the poison floats in the atmosphere at a considerable distance from the original source. It adheres with considerable tenacity to the walls, floors, bedstead, and articles of furniture, but especially to bedding, carpets, curtains, and woolen goods of all kinds. Not all who come in contact with the germ or poison have diphtheria, for individual susceptibility and predisposition are important factors. When the predisposition exists, and exposure is effected, a certain interval elapses before there are any objective signs of the disease. This *period of incubation* is very variable, and the variations are due to the differences in the intensity of the poison and the systemic state of those poisoned. The more malignant the disease and the more depraved the bodily condition, the more quickly will the symptoms of the disease appear after reception of the disease-germs. If the poison come in contact with an abraded surface, it secures immediate admission to the blood, and then the stage of incubation may not exceed two days. Admitted to the system in the ordinary way, the period of incubation will vary from three to ten days. By Oertel it is placed at two to five days. According to the author's observations, the period of incubation during the epidemic prevalence of the disease is in the largest number of cases three days.

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* Virchow's "Archiv," Bd. ix, s. 228, 1856.

† "An Account of the Putrid Sore Throat," by John Fothergill, M. D., fifth edition, London, 1769, p. 31.

Pathological Anatomy.—Except for the nicer pathological distinctions of modern methods, we might adopt the description of Fothergill* as an account suitable for to-day of the lesions of diphtheria. The first change consists in hyperæmia—a vivid injection of the mucous membrane of the fauces. At the end of twenty-four hours a faint, grayish-white pellicle appears on the surface of the soft palate, the pillars of the fauces, the pendulum, or the tonsils. The patches may be no larger than pin-heads, and scarcely thick enough to prevent the membrane showing through them. In a few hours they greatly increase in number, coalesce over spaces having the area of three or four lines, and thicken, so that they appear like bits of curds on the surface of the membrane. Now there appear, constituting the exudation and piercing the mucous membrane, forcing apart the epithelial cells, great numbers of round bodies, highly refracting single cells with thick walls—the micrococci. Masses of them, united in bundles and colonies, form distinct nodules, projecting above and making their way into the deepest part of the mucous membrane.† Leucocytes—pus-corpuscles—soon appear, but not in great numbers, in the deep layers of the mucous membrane, and they are coated by micrococci, and these bodies have also penetrated their interior; but, as the process extends, pus-cells increase in number and spread out through the basement membrane and through the epithelial cells surrounding the micrococci colonies on all sides. Among the pus-corpuscles now appear young cells three or four times larger than the former, and they multiply in large numbers—their nuclei surrounded by a thin layer of protoplasm, accumulating also. Thus is formed a mass composed of micrococci, pus-cells, and newly formed cellular elements, which constitute a membranous patch that may be lifted off the surface.[*] In the *croupous form* a quantity of fibrin is exuded when the local process has reached the development above described. This fibrin is poured out into the epithelium, and between the epithelium and the basement membrane or "sub-epithelial tissue." The epithelial cells rapidly undergo necrosis; a network of fibrin develops between them, and colonies of micrococci form in the outer layer of the false membrane. Succeeding exudations lift up the first-formed false membrane, and between them capillary hæmorrhages may take place, and thus the extravasated blood is inclosed in the meshes of the fibrinous exudation. Meanwhile the micrococci penetrate deeper, new deposits of fibrin occur, and hence the false membrane increases in all directions and new ones are formed. The membrane is detached and cast off by a cessation of the fibrin exudation and an abundant formation of pus elements. The micrococci penetrate to the lymphatics and lymph-canals, unless cut off from penetrating below by the abundance of the fibrinous exudation. The mucous membrane of the nose, larynx, and air-passages, undergoes similar changes in the process of formation of a false membrane. When recovery takes place the fibrin exudations cease, and the false membrane is broken up and detached by the abundant formation of merely purulent cells. The epithelium destroyed is restored by the formation of new cells produced from the sub-epithelial layer. In the *septic form* the masses of false membrane undergo decomposition, bacteria form in immense numbers, and the micrococci penetrate to the deepest part of the mucosa, filling in the sub-epithelial and sub-mucous tissues. It is generally conceded that the diphtheritic process as it occurs in the nose is more apt to produce septic infection. Here the micrococci accumulate in the greatest numbers, and seem possessed of the greatest activity; for the periosteum, the cartilages, even the bones, are attacked. *Gangrene* is produced in consequence of the rapid increase in cells, the exudations of fibrin, and the crowding of the tissues by micrococci, arresting the blood-supply and stopping the nutritive processes, hence causing a necrobiosis, which is extensive in proportion to the spread of the membrane formation. When this occurs, "false membrane mucosa, and submucosa form together one semi-liquid, discolored, dark pulp, or a darkish, wormwood-like, broken-down mass, or a dark, more firmly attached slough, from which the intense, peculiar odor of gangrene is spread."[†]

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* Fothergill did not, as Bretonneau points out, properly distinguish the diphtheritic sore-throat of scarlet fever from diphtheria.

† Dr. L. Letzerich, "Beitrage zur Kenntniss der Diphtheritis," Virchow's "Archiv," Band xlvi und xlvii, 1869.

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[*] Burdon-Sanderson long ago described, with his usual fidelity, the fibrin, the cellular elements, and the transparent granules (micrococci ?) which unite to make up the false membrane. ("Contributions to the Pathology of Diphtheritic Sore Throat," etc., "British and Foreign Medico-Chirurgical Review," January, 1860, p. 179, *et seq.*)

[‡] Jaffé, "Die Diphtherie," etc., Schmidt's "Jahrbücher," fünfter Artikel, vol. clviii, p. 73. Also, Oertel, *supra.*

The lymphatics of the neck, whose vessels take their origin in the tissues included in the diphtheritic process, are also involved. The micrococci penetrate to the vasa efferentia, and are seen crowding these vessels in large numbers. The lymphatic glands of the part—submaxillary, sublingual, parotid—and the chain of cervical lymphatics underlying the sterno-cleido-mastoid are enlarged more or less extensively. The periglandular and the general connective tissue are swollen, infiltrated with pus and lymphoid cells, and there may be also around the glands extravasations of blood. The swelling of the glands themselves is due to a hyperplasia of the cells, the stroma remaining unaffected. The membranous exudations, in a small proportion of cases, extends to the bronchi, but only involving a part of the tubes. The changes in the lungs are due to the mechanical obstruction of bronchi, the consequences being atelectasis, emphysema, and localized œdema. When the diphtheritic process invades the lung-tissue itself, there will be seen at various points extravasations of blood, and infarctions, and alveoli distended with cellular elements—epithelium, blood-corpuscles, and new cells, etc.—and micrococci colonies. In cases of septic infection, the muscular tissue of the heart becomes soft, is easily torn, and its fibrillæ are far advanced in fatty degeneration, while at various points are extravasations of blood into the muscular substance. Ulcerative endocarditis, due to the development of bacterian colonies, thickening and vegetations of the valves, with the secondary consequences of this condition of the endocardium, have been repeatedly demonstrated.* The composition of the blood is much altered in the cases of severe toxæmia: it is black, fluid, rather mucilaginous, and stains the fingers a brownish color. Important changes occur in the kidneys, and at a very early period of the disease. They are swollen, intensely hyperæmic in the severe cases, but little so in the mildest; but, in all cases, changes occur in the Malpighian tufts and in the tubules. The tufts are hæmorrhagic, contain micrococci colonies, and are surrounded by lymphoid cells; the epithelium of the tubules is cloudy, granular, and swollen, and is often detached in the form of casts with epithelium adherent. The brain is hyperæmic, and there are numerous capillary hæmorrhages, but the most interesting changes, which serve to explain the secondary paralyses, are those occurring in the spinal nerve-roots, which are thickened, while in the sheaths of the nerves hæmorrhagic extravasations occur, and they are also filled with lymphoid cells and nuclei. Important changes occur in the muscles, beginning at any point of infection. Capillary hæmorrhages † occur in them, and the striæ disappear in the course of a fatty and granular degeneration. Those muscles lying immediately under the affected mucous membrane are apt to undergo these changes, because invaded directly by the pathological products of the diphtheritic process.

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* "Ueber diphtherische Endocarditis," von C. J. Eberth in Zurich, Virchow's "Archiv," Band lvii, s. 228, *et seq.*

† The constant appearance of capillary hæmorrhages, in various parts, referred to in the text, is regarded as highly characteristic. Jaffé, "Die Diphtherie," etc., Schmidt's "Jahrbücher," Band clvii, s. 73. An elaborate article, extending through five issues of the journal.

Symptoms.—There are well-marked forms of diphtheria—the catarrhal, the croupous, the septicæmic, and the gangrenous. In the description of the morbid appearances these natural divisions were kept in view, and all who have had any considerable experience with the disease will recognize the adherence to nature of these phrases. In the *catarrhal form*, the initial symptoms are those of an ordinary catarrh. Heat, irritation, and pain are felt in the throat, and, on the attempt to swallow, much soreness is experienced. Chilliness followed by some slight fever, headache, backache, and general muscular pains are usually present, but in the mildest cases only some slight general *malaise* may result. In still other cases the symptoms may be more pronounced: high fever, severe sore throat, violent headache, *tinnitus*, considerable debility, nausea, and vomiting may be experienced. On examination of the fauces, there are seen more or less intense hyperæmia, and on the palate or tonsils minute grayish-white patches, very thin, and firmly adherent. The tongue is covered with a thick white coating, which extends well forward to the tip, and is also pertinaciously adherent to the organ. In a day or two, sometimes more rapidly, the patches of false membrane extend over the tonsils, the pillars, and the pharynx by a union of numerous centers of deposit, and not by a marginal growth only. The thickness of this membrane is at this time a line or two, and it is distinctly outlined against the dark-red mucous membrane about it. The color of the membrane is grayish-white, but it varies from that shade to dark red, or even black. The reddish tint is due to extravasation of blood, and inclosure of the blood in the meshes of the exuded fibrin. In the catarrhal form, however, but few cases attain to such an extent of false membrane; there are a few patches which may coalesce and be limited to one side, and they reach their maximum by the third day, when already the mucous membrane has become paler, and the exudation is loosening at the margins. The fever which appeared at the outset has by this time disappeared, but in most of the cases of the catarrhal form there is no fever, or it ceases after the first day. The general disturbance ceases with the fever, except the debility, which seems in marked contrast to the apparent severity of the disease. Soreness of the throat, pain in swallowing, and some tumefaction of the submaxillary and deep cervical glands continue up to the detachment of the false membrane, which may take place about the sixth day. When the false membrane is detached, the mucous membrane appears red and still swollen, but its continuity is restored by the production of new epithelium. In the more severe cases the detachment of the false membrane is not effected until some days later, the debility is considerable, and convalescence requires several days longer. The mildest cases of the catarrhal form may be followed by diphtheritic paralyses and other sequelæ.

Croupous Form.—This form may begin as the ordinary catarrhal variety, and continues to the formation of the false membrane, without any indications of a departure from the usual course, until the fourth or fifth day, when it takes on a new character by the sudden development of a high fever, increased tumefaction of the glands, spreading of the false membrane, etc. When the case from the beginning assumes the severity belonging to the croupous form, it sets in with violent symptoms—with chilliness but not a chill, followed by high fever; or the fever begins at once with the onset of other symptoms, the temperature rising to 103°, 104°, or 105° Fahr. The usual symptoms of the feverish state are also present—headache, general pains, thirst, and restlessness at night, occasionally delirium. Then occur the special symptoms referable to the throat—a sense of heat and burning, and severe pain in the act of swallowing. The sublingual and submaxillary glands are swollen, and especially the deep cervical lymphatics lying under the sterno-cleido-mastoid, which are not enlarged in other affections of the throat. The swollen glands are hard and tender, and the infiltrated connective tissue about them is also sensitive to pressure. The mucous membrane is intensely hyperæmic in parts, especially on the pendulum, the palate, the pillars of the fauces, and the tonsils, and it is swollen and œdematous. On this dark-red ground appears, in a few hours, the false membrane in small patches of grayish-white, and, in the course of the next twenty-four hours it has developed into a thick, yellowish-gray membrane, which, becoming drier and darker, presents an appearance not unlike the rind of bacon. In the course of subsequent changes the false membrane assumes a yellowish-gray shade, somewhat like sole-leather. The change in tints is at first due to the inclosure of blood within the meshes of the exuded fibrin, and afterward to the great increase of the pus-corpuscles. If this thick, tenacious, leather-like false membrane is now removed, the epithelium comes with it, leaving a raw, dark-red, bleeding surface beneath. Another false membrane may form on this surface, or it may undergo healing in the mode already described. While the development of the local morbid process is proceeding, the general condition may improve, the fever declining to near normal, the appetite returning, and strength increasing. An arrest of the local process may be effected at the end of the first or beginning of the second week, the membrane become detached, and convalescence be slowly established. More frequently, however, while this apparent improvement is taking place, the false membrane is spreading in all directions. Usually, when no attempt at the arrest of the disease is made, the fever rises higher, the difficulty in swallowing increases, and the patient is tormented by efforts to rid the throat of a tough secretion. At this period of the disease, a condition of profound adynamia may come on, and death ensue in collapse. Otherwise, the disease pursues its course, the false membrane extends, the swelling of the neck increases to formidable proportions, the salivary glands pour out a quantity of offensive saliva, and from the fauces is exhaled a horrible fetor which awakens suspicions of the setting in of gangrene. If the exudation does not extend to the larynx, the breathing, though heavy, is not dyspnœic, and the voice, though muffled and nasal, is not toneless. The appetite is utterly gone, the stomach rather unsettled, although vomiting is not usual, and the bowels are rather constipated, but vomiting and diarrhœa may both exist, caused, it may be, by the swallowing of the ichorous matters produced in the throat. The urine is scanty and high-colored, and in the great majority of cases contains albumen (Squire[*]), and the quantity of urea is increased —at the maximum of the disease, doubled. Casts of the tubules with epithelium, adherent and hyaline cases, have also been observed in the cases of albuminuria. When the disease has reached the point in its development just described, slow recovery may take place, as already mentioned, or the disease may extend into the nares, downward into the larynx and trachea, or into the Eustachian tube. As there are some special features introduced into the symptomatology by such extension of the morbid process, it becomes necessary to enter into brief details on these points. When the membrane spreads into the nose, a disagreeable sense of stuffing is produced, the patient breathes through the mouth, epistaxis frequently occurs, and an ichorous mucopurulent discharge flows from the anterior nares, excoriates the upper lip, and on this raw surface not unfrequently a false membrane forms. This is a serious complication, owing to the fact that septicæmia is very apt to be produced, and death may be caused by profuse epistaxis. The false membrane may spread up the lachrymal duct, and form on the conjunctiva, or, obstructing the flow of tears, cause epiphora. If the false membrane extends into the Eustachian tube, there will occur ear-ache, noises in the ears, deafness, etc. Extension downward into the larynx may take place early in the disease—from the third to the sixth day—or it may not occur until the end of the second week. Laryngeal diphtheria is more apt to occur in young children and in old persons (Oertel). The formation of false membrane may begin in and be limited to the larynx.[†] The capacity of the larynx being greater in adults than it is in children, the symptoms of stenosis are more pronounced in the latter. Progressive difficulty of breathing, a hoarse, then toneless voice, the characteristic "croupy cough," are the symptoms of laryngeal diphtheria. These cases present the clinical history of croup throughout, and the reader is referred to the article on that topic for the details. These cases do not continue very long, and their termination is usually fatal, although recoveries do ensue.* They prove fatal by spasm of the glottis, by obstruction of the bronchi, by pneumonia, by carbonic-acid poisoning, etc. In the rare cases terminating in recovery, the false membrane is expelled by coughing, and no new membrane is produced. The fever and other symptoms subside with the improvement in the local condition.

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[*] Reynolds's "System of Medicine," article "Diphtheria," vol. i, American edition, by Lea.

[†] "Relation of Membranous Croup and Diphtheria," "Medico-Chirurgical Transactions," vol. lii, p. 7. "The evidence before the committee is conclusive as to the fact that in epidemics of diphtheria cases do occur in which the false membrane is thus limited . . . . but such cases are exceptional."

Septic Form.—During the course of the catarrhal or croupous form, especially the latter, the products of decomposition entering the blood, the condition of septicæmia will be produced. The development of the systemic state is preceded by ichorous decomposition of the exudations and secretions of the fauces; a foul-smelling and very irritating fluid is discharged from the mouth; the lips are eroded by it, and on the erosions grayish-white patches of false membrane form. Numerous capillary hæmorrhages occur; the blood mixing with the decomposing membranes gives them a blackish appearance; and the whole mass, putrefying, presents a strong likeness to gangrene, but on removing the decomposing materials the mucous membrane beneath is seen to be merely hyperæmic, and capable of entire restoration. The glands of the neck and the neighboring connective tissue swell enormously, and present a shining appearance, and are hard or doughy to the touch. When the blood is poisoned, the constitution sympathizes profoundly. The face has a sallow, earthy, and pallid hue; the pulse is small, weak, compressible, and very slow; the temperature does not pass above 100°, and is more frequently at 98°, even lower; the appetite is gone, nausea, vomiting, and diarrhœa are usually present, the stools having a foul odor; the urine is small in quantity and loaded with albumen; and the strength is exhausted. Meanwhile the mental condition is that of apathy, the mind acting slowly but correctly, the intelligence becoming clouded only at the last. In other cases, the development of the septicæmia occurring more slowly, the phenomena are virtually the same—the main features being exhaustion, slow and irregular pulse (40 or 50 beats to the minute) or becoming rapid and thready, the temperature below normal (96° or 97° Fahr.), and weakness so great that fainting ensues on attempts to sit up, death usually occurring suddenly from failure of the heart. Recovery, it is claimed (Oertel), has been observed, but death is the usual result in a day or two after the development of the septicæmia, and very rarely later than four or five days after. When recovery is to take place, the pulse gains in volume, force, and frequency, the temperature rises, and the local condition improves. Convalescence is necessarily very slow.

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* "The mortality from this complication is alone very great; it has been estimated that one half of the fatal cases of diphtheria die from this accident" (Squire, *op. cit.*, p. 67).

Gangrenous Form.—This is an extension only of the septicæmic form, and should be so regarded. Gangrene attacks the infiltrated mucous membrane, and the exudations participate in the process. The affected parts turn black, and emit a horrible fetor. Before separation of the sloughs takes place, the blood is poisoned, and the patient rapidly passes into a condition of profound adynamia. Death is produced by thromboses, embolisms, failure of the heart, etc.

Course, Duration, and Termination.—The course and behavior of diphtheria have been sufficiently detailed in the preceding pages. The several forms described are based on sound observation and experience, which must always be confirmed. The mortality of diphtheria varies greatly in different epidemics, and the results of sporadic cases are influenced by numerous causes. In some epidemics nearly all have died. A mortality of one in three, one in seven, and one in ten, has been observed in various English epidemics. So great is the variety in the severity of epidemics and of individual cases, that no precise statement of mortality rates can be made. It is certainly true that no case of diphtheria should be regarded as trifling, for during the course of the simplest cases the most formidable symptoms may arise. The prognosis in any case is the graver, the more virulent the case from which the poison was obtained. The age and constitution of the individual attacked are of moment, for the mortality is much greater in young children, both on account of the danger of laryngeal implication and their feeble powers, and in those of any age who possess poor constitutions, are scrofulous, and enfeebled by bad habits and hygiene. The appearance of successive deposits, the occurrence of albuminuria, and the enlargement of the cervical lymphatics, indicate an extension of the disease. Extension to the larynx, as has already been pointed out, is in the highest degree unfavorable, and especially so in young subjects. Extension to the nasal passages is regarded as very unfavorable, both on account of the greater danger of septic infection and the interference with respiration. Jacobi, of New York, who is high authority, maintains that the unfavorable prognosis of nasal diphtheria heretofore made must be modified, if proper treatment is instituted. Much vomiting and purging are unfavorable symptoms, and in the same way must bleeding be regarded. If the specific gravity of the urine declines, and casts and blood-corpuscles are present, the temperature also rising, these symptoms are unfavorable. If the temperature should rise after the fifth day, it is suggestive of some new development, or of an extension of the exudation. A low temperature, below normal, a cold and clammy skin, and a slow and irregular pulse, are of particularly evil import. Cases that are apparently doing well sometimes terminate very unexpectedly and suddenly by paralysis of the heart. As regards the different forms of diphtheria, the catarrhal is the most hopeful; next the croupous, and lastly the gangrenous. A majority of the catarrhal end in recovery—of the croupous in death.

Sequelæ.—Although the paralyses of diphtheria are really modes of manifestation of the poison, and are referable to changes occurring in nerve and muscle, it will be most convenient to study, together, those which occur during the existence of the other symptoms, and those which appear after the supposed termination of the disease. The latter group of paralyses come on two, three, even six weeks after the healing of the mucous membrane, but the former arise to complicate the case during the second week and subsequently. A nasal tone of voice, some difficulty in swallowing, and the regurgitation of liquids through the nose, are first observed. At length, complete inability to swallow occurs in the third or fourth week. On inspection, the palate is seen to hang limp and lifeless, and no movement is produced by irritation, the sensibility—as Trousseau long ago pointed out—being absent. The power of the heart is greatly reduced at the same period by extension of disease to the motor apparatus. The slowness of the pulse sometimes is phenomenal, the beats descending to 60, 50, 40, and in one case, reported by Sir William Jenner,[*] to 16 per minute. Paralysis of the heart may take place quite unexpectedly, and without any marked change in the ordinary conditions of the circulation. Paralysis of the respiratory muscles may also occur at this period, and may involve the phrenics and diaphragm, as in Sir William Gull's [†] case, or the intercostals and other chest-muscles. There is, probably, no difference, except as to rate of development and severity, between the cases of diphtheritic paralysis occurring in the second week and those which appear as sequelæ. The latter pursue a nearly definite course. They develop slowly but not until after healing of the mucous membrane, and begin in the muscles of the pharynx and soft palate, then involve the ocular muscles, and lastly the upper and lower extremities. These paralyses may follow the mildest as well as the more severe cases. The author saw a fatal case of diphtheritic paralysis of the muscles of respiration in a lady of sixty, who had been treated for a simple sore-throat two weeks before. Donders [‡] mentions the same fact: "Among the cases . . . . there were many in which the angina ran its course without important symptoms, several in which the angina was not recognized as diphtheria," etc. The earliest to appear, and the most usual paralysis, is that of the palatal muscles, causing the voice to assume a nasal tone, and impairing the power of deglutition, especially for liquids, which are regurgitated largely by the nose. Ocular troubles, consisting of dimness of vision, double vision, divergent and convergent strabismus, dilated pupil, disorders of accommodation, etc., are produced by paresis of the third, fourth, and sixth nerves. Shortly after these visual disorders have appeared, numbness, tingling, and pain are felt in the extremities, notably the inferior. These perverted sensations are followed by paresis of the muscles and awkward gait, and ultimately paralysis. The same conditions obtain in the upper extremities—they become paretic, then paralytic. The muscles are apt to waste, and they lose their irritability first to the faradic and finally to the galvanic current, and there is more or less anæsthesia of the plantar surface. Remarkable variations in the extent of the muscular weakness are observed from day to day —a group of muscles not paralyzed to-day may be so to-morrow, and *vice versa.* The muscles of the larynx are attacked not usually at the same time with those of the pharynx, as might be expected, but when there is a wider diffusion of the paralytic symptoms. It may be partial, affecting only one vocal cord, or general, affecting both cords. There may be coincident anæsthesia of the mucous membrane. The voice is hoarse, husky, or wanting; the breathing is troubled if special effort is necessary; and the anæsthesia may permit foreign bodies to enter the glottis, with fatal consequences. Paralysis of the neck-muscles and of the thorax is apt to occur simultaneously, an example of which is reported by Sir William Gull.* When this form of paralysis occurs, the head can not be supported, the respiration is shallow, and the least effort induces dyspnœa. If not soon relieved, the consequences are very serious: the blood is not decarbonized, hypostatic congestion occurs, mucus accumulates, and death happens in asphyxia. The sphincters of the rectum and bladder are usually paralyzed with the lower extremities, and anaphrodisia also is produced. Fortunately, diphtheritic paralysis is very amenable to treatment, and only from five to ten per cent. of the cases prove fatal. A cure is usually effected in a few weeks, but a case of general paralysis may last a number of months. A fatal result is caused by suffocation—the dropping of food into the glottis; by pneumonia, set up by the entrance of some foreign body into the lungs; by failure of respiration; by paralysis of the heart; or by some intercurrent disease.

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[*] "Diphtheria, its Symptoms and Treatment," p. 44.

[†] London "Lancet," vol. ii, 1858, p. 5.

[‡] "On the Anomalies of Accommodation and Refraction of the Eye" (Sydenham Society edition, p. 607).

Diagnosis.—The catarrhal variety of diphtheria may be confounded with acute follicular ulceration of the tonsils, and this mistake is doubtless frequently made. The systemic condition may be much the same in the two diseases, but the local appearances are very different. In the tonsillar affection, there are usually several ulcers at the orifices of as many follicles, depressed below the surface and containing a grayish, cheesy secretion. Pain is limited to the affected tonsil, and the lymphatics under the angle of the jaw are a little swollen and somewhat tender. Both tonsils may be affected when the same conditions obtain on the other side. In diphtheria the exudation is on the surface of the membrane, is not limited to the tonsil, and is accompanied by swelling of the deep cervical lymphatics. The identity or non-identity of croup and diphtheria is still *sub judice.** It seems, however, definitely established that there are cases in which a false membrane is limited to the larynx and trachea, occurring idiopathically and in the proportion of about one to thirty during an epidemic of diphtheria. That a membranous laryngitis can exist quite irrespective of diphtheria is rendered probable by analogy: there are a membranous bronchitis and a membranous enteritis. The fact of its actual occurrence is admitted by Bretonneau, except that he regards it as diphtheria of the larynx. Judged from the clinical standpoint, croup differs from diphtheria in being a local affection, not contagious; the exudation non-specific and formed on the surface of the mucous membrane; in that it does not cause systemic infection, and is not accompanied by albuminuria. The author for these reasons adheres to the non-identity of croup and diphtheria. Between scarlatinal sore-throat and diphtheria close analogies exist, but they may be differentiated by reference to these points: in scarlatina there is an intense and diffused redness of the whole mucous membrane—in diphtheria the redness is merely about the infected area; in scarlatina the exudation is on the surface of both tonsils and usually also on the palate, and is soft like curds—in diphtheria the exudation commences at one or more spots, is attached to the epithelium and is of a grayish-yellow or brownish color; in scarlatina, the symptoms are violent—convulsions, delirium, vomiting, intense fever, inaugurating the disease—in diphtheria the symptoms are not so severe—there are no convulsions, delirium, etc., and only moderate fever; in scarlatina the peculiar rash appears at the end of the first and beginning of the second day, and which desquamates—in diphtheria there is no proper eruption, only transient rashes which are very irregular and accidental.

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* *Supra.*

Treatment.—If the theory of a local infection followed by systemic poisoning be adopted, the early detection and destruction of the first patch of false membrane is of the highest importance. Bretonneau acted up vigorously to the requirements of his theory, and applied muriatic acid to the patches as they appeared. This practice is still pursued by many—by the majority of physicians, probably, but in a modified form. Strong solutions of nitrate of silver; the tincture of the chloride of iron; solution of equal parts of perchloride and glycerine; solutions of salicylic acid, of chloral, of chlorate of potassa, of borax, etc., are those most usually employed. The objections to the use of strong caustic applications seem insurmountable. Experience has shown that the morbid process can not be arrested by the most prompt and efficient applications, for it is impossible to penetrate to all the parts where germs may be deposited; injury done to the healthy mucous membrane invites the spread of the false membrane; the destruction of one layer of false membrane does not prevent the reproduction of successive layers, and it is probable systemic infection takes place during the period of incubation. Those who employ the most powerful applications do not present better results. Cleanliness of the parts, frequent removal of decomposing materials, and disinfection of the discharges, are of great importance for the prevention of septicæmia. These observations are especially true of diphtheria of the nose, the mortality from this being largely due to neglect of cleanliness and disinfection. Oertel * has abandoned and condemns all the strong applications above mentioned, and relies on the vapor of hot water containing a little salt, or chlorate of potassa, as the means for securing cleanliness, disengagement of the false membrane, and for inducing suppuration. The nares should be carefully syringed out every three or four hours with a weak solution of chlorine, chlorate of potassa, carbolic acid, salicylic acid and borax, etc. The solutions must be very weak, and used freely and frequently. With the spray douche a stream of vapor can be nearly constantly kept playing on the parts. Various disinfectant solutions may be used in this way. The author has seen excellent results from the frequent application of a solution of lactic acid—strong enough to taste sour—by means of a mop. A quantity of this may be applied by a large mop to the fauces, and by a syringe to the nares. By what means soever the result is accomplished, careful washing of the affected parts is necessary. Afterward there should be thoroughly dusted over the affected region washed sulphur, which is best accomplished by an insufflator. The good effects of this practice are undoubted, and the explanation is not far to seek. A portion of the sulphur is oxidized, and sulphurous acid produced. The application of lime-water by a method originating in domestic practice is deserving of high commendation. It consists essentially in the inhalation of the vapor, as it arises from the slaking of lime. Some pieces of *freshly burned* lime are put into water, and the vapor is directed to the throat and nose, and inhaled. Above all other topical applications, according to some good authorities, is the atomization of a maximum solution of muriate of quinine, used as often as possible, the spray directed into the fauces. In the case of laryngeal implication, an attempt should be made to dissolve the false membrane by very frequent inhalation of atomized lime-water and lactic acid. Emetics are also used, to effect the mechanical displacement of the membrane. Those acting promptly and producing no after-depression are the most suitable for this purpose, as alum, subsulphate of mercury, sulphate of zinc, ipecac, but not tartar emetic.

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* The facts collected by the committee of the Medico-Chirurgical Society for their "Report on the Relations of Membranous Croup and Diphtheria" are very strong and very ably presented. ("Medico-Chirurgical Transactions," vol. lxii, 1879.)

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* Ziemssen's "Clycopædia," article "Diphtheria," *op. cit.*

The treatment of the systemic condition is equally important with the local. There are two principal indications—to limit the spread of the local disease, and to prevent systemic infection. The author has employed, with apparently great advantage, for the first object, bromide of ammonium (two to fifteen grains every three hours). The bromides are eliminated in large part by the mucous surfaces, especially of the mouth and throat, and thus act locally on the very source of mischief. Acting similarly, and in a high degree efficient, is iodine. In the normal state very decided irritation of the fauces is produced by the iodides. In diphtheria the author prescribes the iodide of ammonium with the bromide for the purpose of effecting a modification of the morbid process in the fauces. To prevent systemic infection it is preferable to administer *liquor iodi compositus*—one to five drops every four hours. Carbolic acid may be given with iodine (℞ Liq. iodi comp. ʒ ij, acid. carbol. ʒ j. M. Sig. One fourth of a drop to two drops in water every four hours). The most efficient of the agents to prevent systemic infection, and at the same time act as a food, is alcohol. There are those who maintain that alcohol is of itself sufficient, if only a large enough quantity can be given. From half an ounce to an ounce every three hours is sometimes administered to infants by the advocates of an exclusively alcoholic treatment. It is certainly good practice to commence with moderate doses of whisky or brandy at the onset of the disorder, and increase them as circumstances demand, as the case progresses. It is certainly surprising to observe the large amount which can be taken by even the tenderest subject. That it is proving beneficial is shown by an improvement in the force, rhythm, and frequency of the pulse, by rise in the temperature if below, by a fall in the temperature if much above normal, and by a change for the better in the general state. Quinine is often given with alcohol for the purpose of support, and as an antipyretic when the temperature is high. The use of quinine by atomization has been briefly referred to. It is questionable whether the good effects apparently produced by this mode of application were due to the systemic or local action of the quinine, for much of that reaching the fauces is swallowed. Not only stimulants and quinine, but nourishing aliments, are required in this disease from the beginning. Milk, beef-essence, egg-nogg, etc., must be given systematically, and when collapse is threatened the intervals between the feedings must be short. Those who have personal charge of a diphtheritic patient, and the physician, need to exercise great circumspection to avoid infection. Several physicians have lost their lives by catching matter from the throat in inspecting the parts, by clearing the canula used in a tracheal fistula, and by making autopsies. Whenever a case occurs in a family, it should be at once isolated. All the dejections, expectorated matters, and utensils used about the patient should be immediately disinfected ; clothing and linens used during the illness should be destroyed ; and the furniture and floors should be washed with chloride-of-zinc solution, papering removed and destroyed, carpets disinfected by heat, etc. The author was personally cognizant of the following facts : A family consisting of father, mother, two children, and a nurse, were put into rooms of a great hotel in Saratoga that had just been vacated by a family returning home, *of whom several were ill with some affection of the throat ;* in a week the little boy became affected with severe diphtheria, was removed to another and a larger room, where he died ; and into this room some new-comers were put the day following the removal of the dead body, without any change in the bed or furniture ! How many more victims we do not know. The paralytic affections of diphtheria require iron and quinine, the phosphates, a generous diet, and a change of air. If they do not yield and get well under these measures, special stimulants of the nervous system are then necessary. Strychnine should be given—hypodermatically if the case is obstinate—and the muscles should be first exercised with the galvanic current, slowly interrupted, and with the faradic current when the contractility of the muscle to the latter has been recovered. When paralysis of the muscles of respiration has occurred, prompt application of these remedies becomes necessary. The pneumogastric, the phrenic, and the intercostal nerves must be galvanized in turn by currents of considerable strength, and the diaphragm should be brought directly within the circuit by poles placed on opposite sides. The question of tracheotomy in laryngeal diphtheria is still *sub judice.* The mortality is so large after this operation, as performed in this country, only as a *dernier ressort,* that there is a growing disinclination to its performance. In France it is performed earlier, with better results. Nevertheless, the successful issue of some apparently desperate cases, such as those of Mr. Lawson and Mr. Pugin Thornton, encourages further efforts in this direction.*

CEREBRO-SPINAL MENINGITIS—CEREBRO-SPINAL FEVER.

Definition.—*Cerebro-spinal fever* is an acute, infectious disease, which prevails as an epidemic, and occurs also in the sporadic form, and is characterized by symptoms of excitation, followed by symptoms of depression of the cerebro-spinal functions, by various forms of eruptions on the skin and by fever of moderate grade—the symptoms being dependent on an inflammation of the membranes of the brain and spinal cord. It has received various designations—as *spotted fever*, *epidemic meningitis* (Stillé), *petechial fever* (G. B. Wood). *Cerebro-spinal meningitis* is the term most generally used, and cerebro-spinal fever is that proposed in the "Nomenclature of Diseases."

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* "Transactions of the Clinical Society," vol. xii, pp. 117, 122, "Cases of Tracheotomy in the Last Stage of Diphtheria—Recovery." For an elaborate discussion of the subject, see Dr. J. Solis Cohen's work on the throat; also, "British Medical Journal," April 10, 1880.

Causes.—Cerebro-spinal meningitis prevails under the most opposite conditions of climate and soil, and at all seasons; but certain parts of the globe have not as yet been visited—Asia, Australia, and Africa, except Algiers, having escaped.* Epidemics appear simultaneously in districts widely separated, under circumstances, as to soil, climate, and hygienical surroundings, the most diverse. While these facts are true, it is also evident that season has some slight influence, not directly, but indirectly, through the changes in habits and modes of life imposed by climate. The disease prevails more during the winter and spring, a fact which is true of the epidemics in this country and other places. Local conditions, good or bad hygiene, or station in life, are without influence in its causation. The disease selects by preference the young, especially young men, but no age and neither sex are exempt. Young recruits, the boys of a boarding-school, children, male and female, under fifteen, are favorite victims, while the disease becomes rapidly less and less frequent after twenty-five. There is probably much truth in Hunt's † observation that this disease "has its favored habitat in cold, damp, and overcrowded tenements, preferring prisons and barracks," as respects its appearance among troops. The author witnessed an epidemic among the boy inmates of a military school, most favorably situated as respects the known hygienic conditions, and there was no extension of the disease in the surrounding rather thickly populated neighborhood. "In April, 1863, four cases occurred in a single tent of the Twenty-second North Carolina: three of these cases died, all being from one family of conscripts, while the fourth tent-mate, an old soldier, recovered. It is difficult to define any special circumstances affecting this tent in preference to the others," says Dr. Robinson, who reports the incident. A great many examples have now been collected of outbreaks within very limited areas, as in jails, prisons, individual houses, confined to such areas, while simultaneously similar outbreaks are occurring at distant points. It is supposed that the places visited are in a bad hygienic state, but there must be some other element present, for the nurture and development of which evil hygienic influences are necessary. There must be a peculiar miasm, *materies*

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* Lombard, "Traité de Climatologie Médicale," *op. cit.*, vol. iv.

† "United States Sanitary Commission Memoirs," edited by Flint, chap. ii, on "Cerebro-spinal Meningitis," by Dr. Sanford B. Hunt, p. 383.

morbi, or germ present. The nature of this unknown principle has not as yet been ascertained. The etiological facts thus far presented demonstrate that the disease is not contagious in the proper meaning of the term. That it is infectious there can be no reasonable doubt. Dr. Burdon-Sanderson * concludes that it is not contagious; that there were no instances of spread from the family first attacked; that the disease appeared simultaneously in the two districts, which were thirty miles apart; that in no instance were two persons attacked in one house. Dr. Lidell † says that "no relation by contact whatever can be traced between them," in the cases occurring in Stanton Hospital. The general experience of American physicians, as collected by Stillé, ‡ is against contagion, in the sense that small-pox is contagious.

Pathological Anatomy.—The changes wrought by this disease are almost as distinctive as those of typhoid fever. They are chiefly in the cerebro-spinal axis. The skin after death presents traces of the herpetic eruptions which are usually seen during life. There are extensive suggillations, not confined to the dependent parts only, and large patches of ecchymoses, the body in some instances being almost black (Stillé). The *post-mortem* rigidity is strongly marked, the muscles, in cases that have continued for many weeks, being much emaciated. Besides emaciation the muscles are found to have undergone granular degeneration to a greater or less extent. The dura mater and arachnoid may be but little altered, but usually present traces of hyperæmia, the arachnoid rough and opaque also. The pia mater is always congested, often intensely punctated with capillary hæmorrhages, and thick and opaque by reason of interstitial exudations. After the initial hyperæmia, wandering leucocytes in great numbers are found in the neighborhood of the vessels, and these are the only changes seen in the fulminant form, because there has not been sufficient time to develop others. After a day or two, the subarachnoid spaces contain more or less cloudy serum, sometimes reddish from the presence of red blood-corpuscles. Next, the membrane is infiltrated by an exudation composed for the most part of purulent elements having a greenish or yellowish color; the exudation may be more consistent, firmer, and of a gelatinous character. Dr. Burdon-Sanderson found that the gelatinous material consisted of cells having many points of resemblance to but still differing from pus-corpuscles, and that the interstitial substance was crowded with granules. The exudation may be several lines in thickness, and it is found in greatest abundance along the great vessels in the fissure of Sylvius, about the optic chiasm, infundibulum, pons, and cerebellum. The whole convexity of the hemispheres may be covered, but usually here the exudation is most abundant in the sulci between the convolutions. As regards the visceral arachnoid, which is usually more or less thickened and opaque, Klebs * has found that this change is due to purulent infiltration. Similar structural alterations are found in the membranes of the spinal canal. The dura mater sometimes presents the same character of changes as in recent pachymeningitis (Klebs, s. 333), at least the hæmorrhagic extravasation; the arachnoid is more or less cloudy from infiltration with pus-cells; but the most important of the alterations are those in the pia, which is strongly adherent to the cord at all points. As in the brain, the first morbid appearance consists in hyperæmia, and then serum, pus, gelatinous exudation of greater or less thickness, the nerve-roots entirely covered with a thick layer of exudation, follow in order according to the time given to them. It follows, then, that in the fulminant form, death occurring in a few hours, there may be but little evidence in the spinal canal of the severity of the malady. The ravages of this disease are not limited to the membranes. The ventricles contain more or less turbid serum, the ependyma and the choroid plexus are hyperæmic, and there may be more or less of the purulent exudation. Those portions of the brain and spinal substance adjacent to the pia mater are, in advanced cases, altered by hyperæmia and by the imbibition of fluids, so that the nerve-elements are more or less dissassociated (Klebs). In cases of long standing, the effusion may be so great as to cause flattening of the convolutions and œdema of the brain. In one case the central canal of the cord was filled with pure pus (Ziemssen). Besides these *post-mortem* appearances which are necessary to constitute the disease, various alterations have been found, and some of them so constantly as to justify the opinion that they are parts of the morbid complexus. The heart-muscle, as it is in other fevers, is soft, friable, and granular in the cases of some weeks' duration, but unaltered in the fulminant form. The blood is dark, fluid, wanting in coagulability, and the walls of the vessels are stained by it. The lungs frequently present evidences of bronchitis, catarrhal pneumonia, atelectasis, etc. The hepatic cells and the tubular epithelium are cloudy and more or less granular by deposit of fat-granules (Klebs), a change which is likened to that which takes place in phosphorus-poisoning.

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* "Official Report on the Epidemic of Cerebro-spinal Meningitis of Northern Germany," London, 1865.

† "American Journal of the Medical Sciences," January, 1865, p. 1, vol. xlix.

‡ "Epidemic Meningitis; or, Cerebro-spinal Meningitis," by Alfred Stillé, M. D., Philadelphia, Lindsay & Blakiston, 1867, p. 178.

Symptoms.—There are marked differences in the behavior of cases of cerebro-spinal meningitis, but they may be comprehended in four groups: the ordinary or common form; the fulminant; the petechial; and the abortive.

The Common Form.—Almost always the disease begins abruptly, and if prodromes exist they are headache, muscular pains, vertigo, and fatigue, which disappear just as the disease is about to manifest itself.* A chill, or a decided sense of chilliness, an intolerable headache, nausea, vomiting, vertigo, and an overwhelming sense of weakness and illness, are the formidable symptoms with which the disease opens. The pain in the head may be like a constricting band, especially about the forehead, or a boring or lancinating pain shooting in all directions, or the whole head is the seat of an intense but indescribable anguish. With every attempt to rise up, vertigo comes on and vomiting is induced, but when recumbent the vertigo often persists, the patient seizing hold of the bed to keep steady. The vomiting is causeless so far as the stomach is concerned; at first food and afterward some mucus and bilious matter come up. In a few hours the muscles of the neck become somewhat stiff, and pain is experienced with every attempt to turn the head. An extension of this state of the cervical muscles takes place to the muscles of the spinal column, which become stiff, rigid, and painful with all attempts to move the body. The muscles of both upper and lower extremities are affected in the same way, and the motions of flexion and extension are both painful and awkwardly performed. At the same time symptoms of irritation of sensory nerves are experienced. The surface of the body generally is highly sensitive, but the skin of the temples, neck and face is especially so, a light pinch causing expression of suffering even when insensibility is profound. Headache is, however, the source of greatest suffering, which is manifest by restlessness and groaning during the existence of more or less complete insensibility. At the outset high mental excitement introduces delirium; in children, convulsions may occur; the delirium may be active, maniacal, the patient difficult of restraint, or it may assume a busy, trembling character. The symptoms of excitation in the mental sphere do not continue long, for effusion which occurs in the course of the first day causes depression of this function, and the excitement or delirium gives place to somnolence or stupor. The rigidity of the neck increases, and, the spinal muscles also contracting, the head is drawn back and the spine curved; the forearms are partly flexed on the arms, the legs on the thighs. In the milder cases there is a condition of somnolence, from which the patient may be aroused and will answer correctly in part, but he at once falls into stupor, or the state of somnolence is interspersed with paroxysms of active delirium. Besides the condition of rigidity of the muscles generally, attacks of cramp and transient spasms occur. Convulsions at the outset in the case of children have already been referred to, but the cramps and spasms here intended occur in groups of muscles—cramps in the muscles of the legs chiefly, and spasmodic twitchings in the muscles of the lips, eyelids, etc. The face is usually pale and sunken, the features fixed, sometimes retracted (*risus sardonicus*), and always expressive of suffering, manifested in the deepest stupor. The special senses are more or less disordered. Intolerance of light is succeeded by double vision, amblyopia, and, in some cases, amaurosis; tinnitus aurium, vertigo, and intolerance of sounds, are succeeded by impaired hearing, in many cases by permanent deafness. Taste is lost, appetite is absent, and vomiting is frequent. Constipation exists at the first part of the disease, but toward the close diarrhœa and involuntary evacuations occur. The tongue becomes very dry and cracked; sordes accumulate about the teeth, some blood exudes from the gums and nares, and the hardened clots block up the anterior nares and collect about the teeth. It not unfrequently happens that lumbrici are thrown up in vomiting. It is remarkable how little the circulatory system participates in the inflammatory disturbance of the nervous system. The pulse is usually a little quickened, but it does not exceed 100 as a rule within the first four or five days; but very distinctive features are the irregularity of the pulse, the unaccountable quickening, the equally unaccountable slowing, and the variations in tension. The respiration is equally irregular—at first quickened, and afterward becoming variable in respect to the depth and rhythm. When sufficient effusion occurs to compress the medulla—in from three to five days—the respiration assumes the well-known Cheyne-Stokes type—is sighing and irregular. Various kinds of eruption appear on the body, but these are not observed in every epidemic, although it is our observation that some form of eruption will be found if careful search be made. Herpetic eruptions are most frequent, next roseola and urticaria—all eruptions belonging to the group of trophic affections, and petechiæ, those due to disintegration of the blood. The most frequent site of the herpes is on the face, but it may occur on any part, while the others are distributed over the body irregularly. Having attained its maximum in from three to six days, the case may take either of two directions—to a fatal termination; to recovery. In the fatal cases the stupor deepens into profound coma; the symptoms of motor and sensory excitation yield to those of depression; the rigidity and contraction relax; the extremities become limp and paralyzed; the paralysis may be general or limited to one side; the pupils are dilated and motionless, the eyes deeply sunken and surrounded by a dark ring; no noise awakens a reponse; deglutition is slowly and at last not at all performed; the evacuations are involuntary; the temperature rises in some cases to 105°, 106°, even 108°, and the pulse beats too rapidly to be counted In the cases taking the other direction, the symptoms of depression are necessarily slight and transient, for any considerable depression indicates an amount of damage done by the effusion such as to be incompatible with recovery. In the favorable cases the rigidity of the neck and spine gradually subside, but do not entirely disappear for some time after recovery; the vomiting ceases; the headache subsides but does not entirely disappear, and the strength is slowly regained. From the well-marked cases, as just described, to the abortive form, there are numerous gradations in severity. During every epidemic, and also of those occurring sporadically, many of the cases are very mild. In such examples we observe the sudden onset, considerable headache, stiffness of the muscles, but little or no delirium, and no symptoms of depression.

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* "Zur Pathologie der epidemischen Meningitis," von Dr. Klebs in Berlin, Virchow's "Archiv," xxxiv, s. 327, *et seq.*

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* Githens says that "there is a week of prodromata," "American Journal of Medical Sciences," July, 1867, "Notes of Ninety-eight Cases of Epidemic Cerebro-spinal Meningitis," etc., by W. H. H. Githens, M. D.

The Fulminant Form.—In this form we find the poison in its most active condition. The patients are struck down in the midst of full health, and pass in a few hours into a state of collapse. There is usually a severe chill; the patient becomes cyanosed; the skin grows cold, and is covered with a clammy sweat; the face shrinks, and is livid; the eyes, surrounded with black rings, sink deeply in their orbits; intense pain is at first felt in the head, but in a short time consciousness is lost, preceded by brief delirium; respiration is slow, labored, and sighing; the pulse is rapid, feeble, and soon ceases at the wrist; purpuric blotches appear on various parts of the body, which sometimes quickly vesicate and slough; the urine is scanty, and loaded with albumen. Such cases prove fatal in a few hours or in a few days. Fortunately, they have occurred less frequently in the later epidemics, and are rare, if not unknown, in the sporadic form.

The Petechial Form.—This differs from the ordinary form, in the greater tendency which the cases exhibit toward dissolution of the blood. Bleeding takes place from the gums and nares, and extravasations occur under the skin at various points, forming petechiæ and vibices. In the severest cases of this form, the symptoms are extreme from the beginning, there are great prostration, extensive purpuric patches, vibices, and ecchymoses, coma appears early, and a fatal result is reached in three or four days. In the lighter cases, the only departure from the course of the ordinary form is the occurrence of numerous and extensive ecchymoses and vibices, and of hæmorrhages from the mucous surfaces. The mortality has apparently not been the greater by reason of this preponderance of the purpuric spots.

The Abortive Form consists in the occurrence of headache, stiff neck and spine, vomiting, without fever, in those who are much exposed to the disease, as mothers, nurses, physicians, etc., but are not susceptible further than this to the action of the poison. The malady in this abortive form does not require confinement to bed, and ceases, without further development, in two or three days. Writers (Ziemssen) also describe an *intermittent form*, but there are no differences really between this and the ordinary form; for the range of temperature is so irregular that no typical thermal line can be drawn for this disease.

Course, Duration, and Termination.—None of the acute infectious diseases present such irregularities in their course as cerebro-spinal meningitis. From the course of the fulminant form to that of the ordinary form with the usual complications and the protracted convalescence, diversified by relapses, there is an enormous difference in point of duration. While the former occupies from four or five hours to two or three days, the latter continues four weeks, six weeks, three months, according to the behavior of the several stages. The severe cases of the ordinary form terminate in from one to two weeks. Cases that are very protracted usually terminate in recovery, although various disabilities may remain, but deaths have occurred in the sixth or seventh week (Radcliffe *). The mortality has varied greatly in different epidemics, from eighty to twenty per cent. It seems to be established that the general mortality is declining, rather than increasing, although some recent statistics place it at almost the highest point. In a late Massachusetts epidemic the mortality was a little over sixty-one per cent., and in the last Philadelphia epidemic it was thirty-three per cent. (Stillé). During the same year the mortality at Hardwicke Hospital, Dublin, was eighty per cent. (Radcliffe). The sporadic cases are as a rule much less severe than those during an epidemic. The severity of the disease is increased by various complications, and the recovery hindered by sequelæ. The most important of these complications is broncho-pneumonia and albuminuria. As regards sequelæ, every epidemic leaves behind sad examples of the ravages committed in the brain and organs of sense. One of the most usual cerebral affections left by the disease is chronic hydrocephalus. After the cessation of the inflammation, morbid products, contracting and solidifying, compress the vena Galeni and the straight sinus ; the ependyma undergoes considerable thickening, and the fluid in the ventricles increases. Flattening of the convolutions and atrophy of the brain are the results. In the cases which have come under the author's notice, the head was large, the mind weak, the skull apparently thin, the eyes prominent, the extremities paretic, and the muscular acts incoördinate. Headache is a pretty nearly constant symptom ; but, at intervals which are, however, not regular, paroxysms occur in which intense headache, vomiting, vertigo, and prolonged stupor with delirious intervals occur; sometimes there are convulsions, unconsciousness, and involuntary evacuations, or there may be merely severe headache, intolerance of light and sound, vertigo, and vomiting. If the interval between the seizures is long, considerable improvement may take place in the general health, and expectations of recovery may be entertained. Usually death takes place in one of the seizures, or the patient may be cut off by some intercurrent disease. Recovery very rarely ensues, if possible at all. Partial recovery is not uncommon—the mind being weak, the special senses impaired, members paralyzed and deformed. Paralysis of cranial nerves, hemiplegia, defects of speech, etc., are results of cerebro-spinal meningitis produced by the organization of the exudation, the pseudo-membrane causing injury of parts by pressure. The special senses are very frequently permanently damaged. The eye is injured by a simultaneous suppurative inflammation, and by the extension of the inflammation along the sheath of the optic nerve. Iritis, choroiditis, retinitis, opacities of the cornea, are the most important. The auditory nerve is readily injured, owing to its softness of texture; hence we may suppose the frequency with which impaired hearing results, but inflammation of the internal and middle ear often occurs simultaneously with the inflammation of the meninges. Further, inflammation may extend by contiguity of tissue along the sheath of the auditory nerve. The result is that, in a large proportion of cases, dullness of hearing to deafness is found to exist after the termination of the disease.

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* Dr. J. Netten Radcliffe, Reynolds's "System," article "Cerebro-spinal Meningitis," American edition, H. C. Lea's Son & Co., 1880.

Diagnosis.—Cerebro-spinal meningitis may be confounded with tubercular meningitis and typhoid fever. The distinction between tubercular and epidemic meningitis rests on these points: the former is always sporadic; is preceded by prodromic symptoms; its course is marked by decided crises; the rhythm of the pulse and respiration is much disturbed, and there are no eruptions. As, however, the same tissue is involved and by an analogous process, it need not occasion surprise that these diseases present very similar symptoms. The differentiation from typhoid rests on these points: typhoid comes on more slowly, is without the intense headache, the muscular rigidity, and the causeless vomiting of meningitis; in typhoid there is diarrhœa—in meningitis, constipation; in typhoid there are some hebetude of mind, muttering delirium, stupor—in meningitis, active delirium terminating in coma, or stupor interspersed with delirium; in typhoid there is a typical thermal line—in meningitis there is no regular course to the fever; in typhoid the disease develops slowly to its maximum—in meningitis the maximum is reached in four or five days; in typhoid there is a characteristic rose-colored, lenticular eruption—in meningitis there are various kinds of eruptions, pursuing no definite plan.

Treatment.—The accumulated experience of the medical profession seems now to indicate the superiority of opium as a remedy for cerebro-spinal meningitis. The author has witnessed some striking examples of its value, especially in the form of morphine hypodermatically. In Germany it holds the first place (Ziemssen). In various epidemics, Boudin has found opium the only remedy worthy of confidence. Stillé strongly advocates its employment. There are two points in regard to the administration of opium, on which the author strongly insists—early and efficient administration. It should anticipate the effusion by an antagonistic action on the vessels. To accomplish this object, large doses of morphine are necessary, for, as every observer has witnessed, there are a remarkable increase of the arterial tension and slowing of the heart produced by a full dose ; and these are the conditions most necessary to prevent migration of the white corpuscles. Aside from theoretical considerations, it has been observed that there is a singular tolerance of opium in this disease. A decided effect should be produced, and the quantity necessary must be prescribed. The period when opium or morphine may be most useful is limited by the effusion ; after the first four or five days it is less important, but its utility does not cease until the symptoms of depression come on. Quinine and ergot have both been largely used in this country, with and without opium, but the evidence in favor of these remedies is not satisfactory. If there is active delirium, fluid extract of gelsemium (one to five drops every two to four hours) is useful in allaying excitement. When the period of depression approaches, quinine, carbonate of ammonia, and especially turpentine, which is more particularly indicated when the skin is relaxed and cold, are the most useful remedies. Although ice-bags and cold applications are much advised for the head and spine, the author holds that they do mischief by the depression of the circulation which they cause. He advises instead, the use of hot water applied by a sponge, passed over the spine every two or three hours. If there is constipation, a mercurial purgative may be given, but the best authorities condemn the use of mercury to procure absorption of the inflamed products—a bit of English practice lately revived in Germany. On the other hand, iodide of potassium has been used with success to remove adventitious products after the acute attack has subsided. The success of this measure will be promoted by the frequent application of a hot douche to the spine, flying-blisters, and the passage of a weak, continuous galvanic current, but not until all local disease has wholly subsided. As this disease is marked by great depression of the vital powers, stimulants are needed early, but they should not be given recklessly. When the pulse becomes stronger and more regular under their use, they do good ; but, if the tongue grows dry and the delirium more exciting, they do mischief. A generous diet is required from the outset. Milk, eggs, beef-juice, mutton-broth, etc., should be given every three hours, day and night, to avoid paroxysms of weakness in the early morning.

INFLUENZA—EPIDEMIC CATARRH.

Definition.—*Influenza* is a specific epidemic disease, self-limited, characterized by catarrh of the respiratory organs, and sometimes of the digestive, and by nervous symptoms and debility.

Causes.—Epidemics of influenza have appeared from early in the sixteenth century until the nineteenth. Parkes, however, traces back epidemics to the ninth century. The usual duration of an epidemic is two to four years, during which the whole habitable globe may be visited. An epizoötic, similar in all respects to the epidemic in the human family, has occasionally prevailed as widely among horses. Influenza occurs in all climates and latitudes, and visits on its rounds all countries in both hemispheres; but it may limit its ravages to one hemisphere, or to a single country. The rate of its progress varies: thus Europe has been gone over in six weeks by one epidemic—in six months by another. The rate of spread varies as much in particular countries visited, and a month has been consumed in the extension of the epidemic influence from London to Edinburgh (Parkes). As it prevails under all conditions of soil and climate, and is not contagious, there must be present some morbific principle in the atmosphere. That it is a minute organism is a theory which best explains the facts connected with the spread. During several epidemics it has been observed that various kinds of fungi flourished in unusual abundance. There is no regular period of incubation, but attacks occur immediately on exposure, and in other cases not for some days. One attack does not procure an exemption from future ones.

Pathological Anatomy.—The changes of structure proper to this disease are limited to the broncho-pulmonary mucous membrane. An intense hyperæmia takes place in the nasal, pharyngeal, laryngeal, tracheal, and bronchial mucous membrane. The hyperæmia is usually confined to the larger tubes, but it may extend to the finer tubes, so that atelectasis may be associated with it. Pneumonia, both croupous and catarrhal, are occasional complications. In a portion of the cases the gastro-intestinal mucous membrane is also strongly hyperæmic, and a quantity of watery or thick viscid mucus is produced, but this seems accidental. Doubtless, changes in the blood and in the nervous system, of a very subtile kind, must take place, for those occurring in the respiratory tract are not adequate to explain the nervous symptoms and the evidences of blood-poisoning.

Symptoms.—The onset of the disease is sudden. There may be a decided chill, or chilliness alternating with flushing and heat, and fever at once comes on, soon rising to the maximum, but in other cases the febrile symptoms develop slowly, and do not attain their maximum until two, three, or even four days. The course of the fever is remittent, the exacerbations occurring at night. With the rise of temperature there is an increase in the pulse, the number of beats approximating 100. At the same time a severe headache, located in the frontal sinuses and extending into the eyes, is experienced. Soon after the rise of temperature, in respect to which all observers are agreed, the symptoms of an acute catarrh come on: there occur heat, stuffing, dryness, quickly followed by increased secretion, and sometimes epistaxis; the conjunctivæ are injected, and the eyes are watery; presently the throat feels hot, dry, and irritated, and spots like measles are to be seen on the palate; the mucous membrane of the mouth and tongue are also hyperæmic, but less so than the fauces. Soon the voice grows husky; a troublesome cough, and, after a time, abundant thin, acrid mucus, and afterward purulent expectoration, are brought up, but at first the cough is hard, dry, and tormenting, especially in the evening and at night, and occasionally vomiting is excited by it. At first there is almost incessant sneezing, but this subsides as the secretions increase. As the catarrh descends into the respiratory organs, the symptoms grow more serious. The expectoration may become bloody; more or less dyspnœa is experienced by many, and sharp stitches are felt in the sides; sibilant and sonorous *râles* are audible over the tubes, and the signs, rational and physical, of pneumonia or pleuritis may be added to those of the disease proper. Instead of this gradual progression of the symptoms from above downward, the nasal, pharyngeal, laryngeal, and tracheal mucous membrane may be affected simultaneously. In ordinary cases the catarrh reaches its maximum on the second, third, or fourth day, and then declines, ceasing after some days longer. As the symptoms develop along the respiratory tract, in a portion of the cases the gastro-intestinal mucous membrane is affected. At first the œsophagus is attacked, then the membrane below. The appetite is gone, there is a good deal of nausea, and vomiting occurs spontaneously, or is excited by the cough or by the presence of food. The epigastrium is painful and there are colicky pains; sometimes diarrhœa occurs—sometimes there is obstinate constipation. A remarkable phase of this disease consists in the disturbance of the nervous system, which is quite out of proportion to the gravity of the local disease or to the amount of fever. From the beginning the patients appear anxious and depressed, and are weak, unequal to any exertion, and confused by any attempt at mental effort. There are general muscular pains and soreness, flying pains along the course of the principal nerve-trunks, but the chief source of suffering is the frontal headache. Besides the hebetude of mind observed to a less or greater extent in all cases, there is sometimes delirium; in still other cases a remarkable state of somnolence has been noted. Vertigo is present in most of the cases; and in some there is a decided hyperæsthesia of the skin of the head and neck. Sweating is not usual at first, and if it occur soon is significant of an early subsidence of the fever, but it is one of the critical phenomena marking the termination of the disease. When there is much sweating, sudamina are present. The urine is usually lessened in amount and sometimes scanty or suppressed. The sweat is said to be highly acid, and the urine also acid and high-colored.

Course, Duration, and Termination.—There are great variations in the intensity of epidemics and of individual cases. Some races suffer severely, others slightly. Children are less susceptible, and have the disease more mildly. The weak and cachectic and the aged run greater risks than the robust and young. Uncomplicated cases pursue their course in from four to eight days; the fever reaches its maximum on the third, fourth, or fifth day, and then terminates by crisis or by lysis. The critical phenomena consist in a profuse sweat, a free urinary discharge, an attack of diarrhœa, or an epistaxis. In the cases declining by lysis, several days are occupied in the return to the normal state. Relapses are by no means uncommon. Cough and expectoration due to bronchitis may persist for some time after the disease; the nervous symptoms may linger and delay convalescence, or complications may arise, or sequelæ follow after the disease proper. Capillary bronchitis and catarrhal pneumonia may result by an extension of the morbid process from the bronchial tubes. A severe conjunctivitis, tonsillitis, or laryngitis, may develop from the usual implication of these parts. Besides these diseases, which are merely exaggerations of ordinary lesions, existing maladies may be much aggravated by an influenza. Those so affected are especially phthisis, emphysema, dilated heart. Pregnant women attacked with influenza are apt to abort. Notwithstanding its apparently profound impression on the organism of man, the poison of influenza is scarcely lethal. The mortality of the last epidemics has not exceeded two per cent. where the disease appeared most noxious. Fatal cases, when they occur, seem to be due to the complications which arise in the course of them or to the sequelæ.

Treatment.—Repose in-doors, a generous diet, and the moderate use of stimulants, are the most important measures. At the outset a full dose of quinine and morphine (gr. xv—gr. ss.) exercises a favorable influence; and throughout the disease these are the most useful remedies to quiet the harassing cough and to maintain the strength. If there is much secretion, cocaine, locally, and atropine, may be combined with the morphine and quinine. If the bronchial mucous membrane is severely attacked, small doses of tartar emetic, or ipecac and morphine, are useful (℞ Ext. ipecac fl. ʒ ij, tinct. opii deodor. ʒ iv, tinct. aconiti rad. ʒ i. M. Sig. Six to ten drops every two hours). If the finer tubes are involved, the preparations of ammonia, the iodide, muriate, and carbonate of ammonia, should be freely administered. If the stomach is very irritable, as is the case in many epidemics, the most useful remedies are oxalate of cerium, hydrocyanic acid, minute doses of morphine subcutaneously, carbolic acid, with or without bismuth, etc. For the violent head symptoms which sometimes ensue, the most appropriate remedies are bromide of potassium, gelsemium, duboisine, morphine subcutaneously, etc. If there is much local distress, the vapor of hot water should be sedulously inhaled. When the first irritation is felt in the nares, a solution of hydrochlorate of quinine should be applied and allowed to pass through into the fauces, after the manner of Helmholtz. It is probable that pilocarpine will be found extremely useful in cases of influenza, administered at the outset with the view to abort the malady. Pellets of cocaine placed in the nares, and allowed to dissolve slowly and pass along the cavities posteriorly, have proved to be an effective application.

HAY-FEVER—SUMMER CATARRH.

Definition.—*Hay-fever* is an acute catarrh of the upper air-passages chiefly, occurring at a fixed period annually, and disappearing after a variable duration. It has received a variety of designations besides those above given, as *hay-asthma*, *rose-cold*, *June cold*, *autumnal catarrh*, etc.

Causes.—Those who suffer from an annual visit of hay-fever refer their malady to a variety of causes, and it is probable that various kinds of emanations excite the disease. It is an interesting fact that three members of our profession, themselves sufferers from the disease, have made the most important contributions * to our knowledge of this affection. By Dr. Bostock the disease was supposed to be of a specific nature, and he rejected, from the point of view of his own experience, the supposed agency of emanations from new-mown hay or grasses. Wyman was unable to come to any conclusion in regard to the supposed agency of minute organisms, whether animal or vegetable, but he has carefully indicated the geographical position of the hay-fever zones in this country. Wyman's attention was directed to autumnal catarrh, as this is the form from which he suffered; on the other hand, Bostock recognized the disease as it occurs in June. Probably the most important investigation ever undertaken is that of Blackley, who has shown that the pollen of rye produced the most violent symptoms of hay-asthma, notably sneezing, profuse catarrh, and oppressed breathing, and that the pollen of grasses, of wheat, oats, and barley was, next to rye, the most active in causing catarrhal symptoms. Further experiments showed that the pollen in the atmosphere consisted in the large proportion of ninety-five per cent. of that from the grasses—the *graminaceæ*. That these observations and the conclusions based on them are correct, can hardly be denied. But it is probable that other influences are also necessary. Beard has lately published a monograph * based on a study of two hundred cases, from which it appears that there are several factors concerned in the production of this singular malady. He concludes that hay-fever is essentially a neurosis; that the same form of disease occurs in the spring, summer, and fall; that it is hereditary, and a product of modern civilization, and that, when the predisposition exists, various exciting causes may develop the disease. We believe that these propositions are correct. When the neurotic temperament is present, and a special tendency exists, various exciting causes, as heat, dust, but especially the pollen of grasses, of rye, corn, and rag-weed, may excite summer catarrh. Various cases have been published, showing that a mental impression may excite the disease. Phœbus mentions a case in which the symptoms of hay-fever were excited in a susceptible patient by looking at a highly realistic picture of a hay-field.

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* "Autumnal Catarrh (Hay-Fever)," by Morrill Wyman, M. D., 1872; "Experimental Researches on the Cause and Nature of Catarrhus Æstivus (Hay-Fever or Hay-Asthma)," by Charles H. Blackley, London, 1873, second edition, London, 1880. The disease was first described by Dr. Bostock, giving his own case, "Medico-Chirurgical Transactions," vol. x, part 1, p. 161; also ibid., vol. xiv, p. 437, "On Catarrhus Æstivus."

Symptoms.—There are two forms in which the disease manifests itself—*the catarrhal* and *the asthmatic*—but they are often united in the same individual. Hay-fever is distinctly periodical; it occurs at certain seasons only, which differ in different cases; and, in many persons, it comes on with unfailing promptitude on a certain day. Whether it occur in the spring, summer, or fall, its clinical features are the same.

Catarrhal Form.—There may be warnings of the approach of the disease in a sense of lassitude and weariness, inaptitude for exertion, loss of appetite, a coated tongue, diarrhœa, or constipation, etc., but in a great majority of cases the onset is sudden. In the enjoyment of the usual health, the first symptoms are felt, although it is true those who have had the disease for years know full well the time of its approach, and probably experience various subjective symptoms, which are purely mental in origin. The first symptom is an itching of the eyes, nose, behind the posterior nares, and the palate. This is followed by the flow of a transparent serous fluid, and then sneezing begins, which is most aptly described by Henry Ward Beecher, himself a sufferer from the disease: "You never before even suspected what it really was to sneeze. If the door is open, you sneeze. If a pane of glass is gone, you sneeze. If you look into the sunshine, you sneeze. If you sneeze once, you sneeze twenty times. It is riot of sneezes. First a single one, like a leader in a flock of sheep, bolts over; and then, in spite of all you can do, the whole flock, fifty by count, come dashing over in twos, in fives, in bunches of twenty." The eyes water, and the conjunctiva reddens; the nasal mucous membrane swells and becomes hyperæmic; and so great is the swelling in many instances that the two sides of the passageway approximate, and breathing is then carried on by the mouth. When the swelling occurs, the sneezing is less persistent, or ceases altogether; the discharge which was clear and watery becomes yellowish and thicker, or it may be reddish from an admixture with blood. A very unpleasant sense of heat and burning is felt about the nose and eyes, and pain, which is rather lancinating, shoots through the orbits and frontal sinuses, and sometimes into the head. The throat is hot, dry, and somewhat swollen, and, in consequence of extension of the swelling to the orifices of the Eustachian tubes, the hearing becomes obtuse, and pain sometimes is felt in the ear.

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* "Hay-Fever or Summer-Catarrh: its Nature and Treatment," New York, Harper & Brothers, 1876, pp. 266.

Asthmatic Form.—This begins at the same time, and runs its course with the catarrhal form, or, after an uncertain period, succeeds to the latter. In either case an extension of the morbid process takes place to the larynx and bronchial tubes, which become swollen and hyperæmic; a hoarse laryngeal (croupy) or a wheezy bronchial cough occurs, and asthmatic difficulty of breathing is experienced in varying degrees of severity, from a mere sense of constriction to extreme dyspnœa. In the worst cases the same phenomena are exhibited as in the severe cases of asthma: the patient is unable to lie down, struggles for breath, is pale, and covered with a cold sweat. Remissions occur, but the difficulty of breathing does not entirely cease until the hay-fever is over, and in some subjects more or less bronchitis, with occasional dyspnœa, persists for two or three months afterward. Very alarming symptoms may arise from an extension of the disease to the finer bronchi (capillary bronchitis), or congestion of the lungs may unexpectedly occur, or an attack of pneumonia supervene. Unless some of these secondary diseases happen, the constitutional symptoms are by no means severe. The pulse is a little accelerated, the temperature slightly, if at all, elevated, except during and for a short time subsequent to the asthmatic attacks. The strength is somewhat reduced, the appetite is rather poor, and the discomfort sufficient to render a patient miserable.

Course, Duration, and Termination.—The disease behaves in a definite manner in all cases, and comes on and goes off with the strictest regularity. The duration of individual cases is from a few days to three months, the average being about six weeks. As locality is an important element in the causation, the behavior of cases is much affected by the surrounding conditions. As a rule, if the patient remain at the same place, the violence of the attacks rather increases year by year. Those at first assuming a merely catarrhal form, after a time become asthmatic, and in some instances the author has known the asthma to become a chronic condition, and to occur throughout the year. On the other hand, timely removal from the hay-fever zone may entirely prevent seizures. Although hay-fever never proves fatal, and usually leaves no sign, it may lead to the development of more serious ailments, as asthma, chronic bronchitis, impaired hearing, etc.

Treatment.—For those who possess the means to travel, there is no remedy so effectual as removal from the hay-fever zone in time to prevent the attack. A sea-voyage, so arranged that the patient is on the ocean at the time of the attack, or residence in Europe, especially in Switzerland, during the same period, is always effectual. There are various parts of the United States where exemption from the seizures may also be secured for one or many years, but the immunity does not always continue indefinitely. The White Mountains, the Catskills, the highest points of the Alleghanies, the Adirondacks, and the Rocky Mountains, are to be recommended. Many seashore places can be resorted to with confidence of relief, so long as the breezes blow from the ocean: Fire Island and the Isles of Shoals are among the most desirable. Certain parts of Canada, Mackinaw, and Marquette, on the upper lakes, are suitable resorts for many cases. As no specific has been discovered, the remedies are very numerous. As is the case in the neuroses, a remedy acting favorably on one occasion will usually fail to relieve when employed again. Quinine has been more useful than any other agent, and may be depended on to give more or less relief if used efficiently. Before the access of the paroxysm it should be administered in the quantity of five grains three times a day for a week, and, when the first symptoms of irritation of the nares are felt, a solution of the muriate (the most soluble salt) should be applied to the nares. When the disease has begun, the best results are obtained from full doses of the iodide of potassium—fifteen grains every four to eight hours. If an abundant secretion is poured out, atropine will be found highly useful. The author has had excellent results from minute doses of morphine and atropine (morphine sulphate gr. $\frac{1}{8}$, atropine sulphate gr. $\frac{1}{200}$) when the paroxysm is well advanced. When asthmatic symptoms are experienced, the most useful remedies are iodides and grindelia. Local applications are, if rightly managed, more efficient than internal remedies. Carbolate of iodine may be applied by the post-nasal syringe thoroughly to the posterior nares, and by the straight syringe through the anterior nares (℞ Acid. carbol. ʒ iij, tinct. iodi ʒ v. M. Sig. Add from one to five minims to a gill of water). A simple expedient consists in vaporizing iodine and cautiously inhaling the vapor through the nares. This may be accomplished by placing a few drops of the tincture in a warm vial. Solutions of chlorate of potash, of chloride of sodium, and of iodide of potassium, properly diluted, are also used with effect by the syringe and douche. Cocaine in solution, or in the form of pellet placed alongside the septum and allowed to dissolve slowly, has proved to be the most effective agent for affording relief. The dose will be $\frac{1}{8}$–$\frac{1}{4}$ gr.

WHOOPING-COUGH—PERTUSSIS.

Definition.—*Whooping-cough* is a specific disease, occurring chiefly in childhood, and once only during life, and characterized by successive forcible expirations, and at their termination by a loud, resounding, sonorous inspiration.

Causes.—Rosenthal has shown that irritation of the internal branch of the superior laryngeal nerve produces relaxation of the diaphragm, spasm of the glottis, and a convulsive expiration—the series of acts which constitute a paroxysm of whooping-cough. Hence, we may conclude that the special exciting cause of this disease is a contagious principle which acts upon the respiratory organs, with special excitation of the filaments of the superior laryngeal nerves. The nature of this principle has hitherto escaped recognition. The morbific material may excite the disease at any age, but it is most common from the first to the seventh year, and it happens in females more frequently than in males. Pertussis occurs among all races and classes, and is more prevalent in winter and spring, although it is encountered at other seasons. As epidemics of whooping-cough sometimes precede, accompany, or follow epidemics of measles, a relationship has been supposed to exist between them; but there is no real foundation for such an opinion. One attack removes the susceptibility to the disease, and it is uncommon for a second attack to occur in the same individual. The period of incubation is, probably, about ten days, but it varies considerably.

Pathological Anatomy.—The only lesions are hyperæmia of the mucous membrane of the nares, pharynx, larynx, bronchial tubes, etc., increased secretion after a preliminary dryness of the membrane, the secretion at first consisting of transparent mucus, afterward becoming more or less purulent, and, when this condition has been reached, the redness of the membrane is succeeded by paleness and anæmia. Various pulmonary and cerebral lesions occur also during the course of whooping-cough, but these are complications not necessary to the disease.

Symptoms.—There are three well-defined stages of the ordinary or common form of the disease—the *catarrhal*, the *spasmodic*, and the *terminal*—and there is a complicated form. The catarrhal stage can not be differentiated from an ordinary catarrh. There occur coryza, more or less cough, and slight fever with evening exacerbation, and morning remission or intermittence, general *malaise* and loss of appetite. After one or two weeks the cough changes its character; it becomes more persistent, and assumes a somewhat spasmodic and paroxysmal character. As a rule gradually, but sometimes suddenly, the characteristic whoop is heard. Then the paroxysms have a distinctive character: the cough consists of a succession of short, rapid, expiratory efforts; the face gets red; the eyes swell and protrude; the body is more and more bent forward in the effort at coughing; then, when the breath is entirely exhausted, a deep, loud, crowing inspiration occurs. During each paroxysm there may be two, three, or more of such efforts, and at the expiration of them the patient brings up a quantity of tenacious, glairy mucus, which is dislodged with difficulty, and is often accompanied by vomiting. In the progress of the case, the expiratory effort is less, the inspiratory is not so long delayed, the secretion becomes less viscid and more purulent, and vomiting occurs less frequently. The peculiar *whoop* or sonorous inspiration is after a time wanting to some of the paroxysms, and ultimately ceases altogether. During the paroxysm, the expiratory effort coincides with a partial occlusion of the glottis, the venous blood accumulates, and more or less cyanosis of the face and head is produced; hæmorrhage may occur from the nose, the ears, rarely from the bronchi, and under the conjunctiva. The frequent collision of the under surface of the tongue with the front teeth excites an ulceration of the frænum and neighboring portion of the tongue. In some cases the sudden compression of the abdominal organs, produced by the coughing, gives rise to the formation of hernia, to prolapse of the bowel, and to involuntary evacuations. The duration of the paroxysms varies from a few seconds to several minutes, and the number of them, daily, is very various, ranging from ten to a hundred, the average being about twenty or thirty. During the period of maximum severity, the attacks are rather more numerous by night than by day, destroying sleep, which may ultimately induce a serious state. The frequent vomiting, also, causes such a loss of aliment that considerable weakness and emaciation result. On the other hand, when the paroxysms are widely separated, the health may be fairly well maintained. The action of the heart is very rapid during the paroxysm, but in the interval it may be normal, unless the system is reduced. The skin is more or less relaxed, and during a paroxysm may be covered with sweat. Attacks are induced by various causes. Imitation is a strong motive; the presence of food in the stomach and the inhalation of dust or irritating fumes of any kind may excite attacks. When the paroxysm is about to approach, the child takes refuge with its nurse, or seizes hold of some object of support, the face turns pale, and then comes the explosion.

Course, Duration, and Termination.—In a well-defined case of the ordinary form the course is tolerably uniform. The catarrhal stage continues two or three weeks, the spasmodic three or four, and the terminal stage a week or two, although it may be prolonged by a cough of habit. The course of whooping-cough may, however, be much modified by the occurrence of *complications*. These occur chiefly in the lungs and the brain. In every severe case of whooping-cough there is probably more or less pulmonary congestion, due to the interference with the respiration occasioned by the paroxysms of coughing. When this occurs the breathing is more or less oppressed in the intervals between the paroxysms; the face is constantly somewhat cyanosed; the action of the heart is quick; the pulse is weak, and the general condition is depressed. A frequent and very fatal complication of whooping-cough is capillary bronchitis, with the attendant accidents of atelectasis and broncho-pneumonia. Not unfrequently these complications lead to caseous pneumonia, emphysema, dilated bronchi, and phthisis. If capillary bronchitis comes on, the greatly diminished aëration of the blood increases the passive cerebral congestion, and becomes, therefore, a cause of convulsions in children. The cerebral complications consist in convulsions and hydrocephalus, the result, chiefly, of the mechanical obstacles in the course of the circulation. The fluid is poured out in the ventricles, in the perivascular lymph-spaces, and in the subarachnoid spaces, and the brain is more or less compressed and anæmic. Sometimes a vessel yields under the increased pressure in coughing, and cerebral hæmorrhage results. These cerebral states are accompanied by the usual signs and symptoms. The duration and termination of a complicated case will, of course, be determined by the character of the complication. The usual termination of uncomplicated cases is in recovery, but there are exceptions to this statement. In young and feeble subjects, the action of the heart may be suspended by the expiratory effort in coughing, or exhaustion may result from loss of sleep and uncontrollable vomiting.

Treatment.—Arising from the action of a morbific principle, whose nature is unknown, obviously no cure will be discovered until the nature of the cause is ascertained. The treatment must therefore be merely symptomatic. During the catarrhal stage, those remedies are employed that have been most useful in ordinary bronchial catarrh (℞ Syrup. scillæ comp. ℥ j, tinct. aconiti rad. ♏ xvi, tinct. opii deodor. ♏. viij, syrup. tolu ʒ vij, aq. lauro-cerasi ℥ j. M. Sig. A teaspoonful every two, three, or four hours). Other formulæ may be found under the head of "bronchial catarrh." The iodide and bromide of ammonium given together are highly beneficial during the catarrhal stage and as the spasmodic stage is about to develop. Tincture of aconite-root, tincture of belladonna, deodorized tincture of opium, and fluid extract of ipecacuanha, in suitable proportions according to age, is a most serviceable combination. Tincture of lobelia may be substituted for ipecac in the above formula, as advised by Ringer, who regards it as highly serviceable in whooping-cough. If the child is old enough, a gargle of bromide of potassium may also be used with advantage during this stage. As the spasmodic stage approaches, the antispasmodic remedies come into use. Probably the most efficient of them all is opium, in the form of the alkaloid codeine, which can be employed with proper precautions, even in the case of infants. A slight hypnotic effect should be maintained constantly, if we would obtain the best results from it. The bromides have an undoubtedly good effect in moderating the violence of the spasmodic attacks. Of these, the monobromide of camphor seems on the whole to be most beneficial. It can be given in an emulsion or pill-form, in from two to ten grains, every four hours. The very best results, and often an immediate arrest of the disease, can be procured by full doses of quinine. Not all cases are affected so favorably ; but in the author's experience no single remedy does so much good in this disease. Atropine often acts most favorably, but is uncertain. Cocaine locally and by the stomach is an effective remedy, and although acting similarly to atropine is a far more valuable remedy. The cough by habit, which remains after the subsidence of the paroxysms, is often admirably relieved by dilute hydrocyanic acid. This is also a useful remedy during the maximum of the disease. Excellent results have been obtained from the use of the mineral acids, especially nitric, in the treatment of the disease during its various stages. The acids should be well diluted, and given in some simple sirup, especially as large doses are necessary. Among the more recent remedies are quebracho and grindelia, which are respiratory sedatives, and are often highly serviceable. Some of the so-called mineral tonics—copper, zinc, and lead—have been administered with alleged success. Of these, probably, the best results have been obtained from acetate of lead, which is exhibited in from one fourth of a grain up to five grains, according to the age. If the tubes are much obstructed by mucus, or if capillary bronchitis supervene, emetics may become imperatively necessary. The yellow subsulphate of mercury, alum, apomorphine, and ipecac, are the emetics best suited to the purpose. Good results are obtained by the inhalation of carbolic spray in many cases. An atomizer may be used directly to deliver the spray in the fauces, or indirectly by filling the air of the apartment. A one per cent. solution is strong enough for this purpose. Like other neuroses, whooping-cough is much influenced by psychical impressions. Change of air and scene is therefore highly beneficial. To this mental impression must be referred the supposed agency of the ammoniacal odors of gas-works, and of such medicines as cochineal, which affect the mind by a brilliant color or disagreeable odor.

PAROTIDITIS—MUMPS.

Definition.—*Mumps* is a specific inflammation of the parotid gland, propagated by a peculiar miasm, self-limited, occurring usually as an epidemic, and characterized by a tendency to migrate into the mamma or testes.

Causes.—Nothing is known of the *materies morbi* which give rise to this disease, except their effects. In from five to twenty days after exposure of a healthy person to the atmosphere about an individual having the "mumps," the former is also attacked. It occurs most frequently in males, but also attacks females, and the usual age is from five to fifteen ; but, during the war of the rebellion, large numbers of raw recruits were affected, whose average age was not less than twenty. Like other diseases of the same class, it usually occurs but once in the same individual.

Symptoms.—There is an initial or prodromic period, which may be so slight as to escape observation. It begins with chilliness, general *malaise*, sometimes vomiting, and a fever comes on immediately, with the usual signs and symptoms of that state. In from twelve to thirty-six hours an acute pain is felt behind the angle of the jaw, and penetrates to the throat, frequently into the ear. The jaw becomes stiff, and a swelling appears immediately under the ear and extends forward and upward, forming an immense protuberance in front of the ear and behind and beneath the angle of the jaw. To the touch, doughy and elastic, it does not pit, and is very sensitive. It is usually confined to the parotid gland, but in severe cases, as seen in the army, the neighboring glands are implicated, and an enormous swelling, reaching as low as the sternum, results. In the ordinary cases the maximum enlargement is reached in from three to six days, remains stationary for one or two days, and then rapidly subsides, completing the revolution in from eight to twelve days. In some cases the swollen part becomes intensely red, the color disappearing on pressure, to return immediately after the pressure is removed, and the epidermis desquamating as the swelling subsides. In consequence of the swelling, which often extends to and involves the neighboring tonsil, and the pain produced by all movements of the jaw, there is much difficulty in mastication and deglutition. When sapid substances, especially acids, are taken into the mouth, an acute pain shoots through the cheek into the swollen gland and ear. Speech is also more or less painful and difficult, and the voice is muffled and indistinct. A viscid saliva continuously flows from the partly-open mouth. Often only one parotid is affected, and the other is attacked in a day or two, but it not unfrequently happens that several years elapse before the second gland is infected. A so-called *metastasis* not unfrequently takes place, of which the author has seen a number of examples. During the existence of the parotid swelling, the corresponding testicle becomes painful and swollen, and often a slight bruising of the organ invites the disease. Sometimes the swelling abandons the parotid, when the testis begins to enlarge. This seems like a true metastasis. The mamma, labia majora, and the uterus, are the organs in the female to which the disease is "translated"; but such an accident must be excessively rare. In some instances an interval of several hours occurs between the disappearance from the parotid and the appearance elsewhere, with the effect to produce alarming symptoms of depression, anxiety, almost of collapse.

Course, Duration, and Termination.—The course of the disease is much affected by the hygienic surroundings of the patient and by the constitutional state of those attacked. During the late war, the cases of mumps were accompanied by high fever, often delirium, and by great depression of the vital powers ; pneumonia was a not unfrequent complication, and those who recovered had a tedious convalescence, the blood being much impoverished and the body emaciated. Under ordinary circumstances, mumps is a mild disease, which always terminates in recovery, its duration varying from four to ten or twelve days. The importance of mumps is to be regarded from another point of view. In some persons, the subjects of a dyscrasia, the morbid condition is awakened from its dormant state by an attack of mumps. The tubercular diathesis is the most common of these. Rarely has the gland suppurated, when attacked by mumps, but suppuration is the usual result when an inflammation of the parotid occurs in the course of typhoid fever. Atrophy is said to have taken place, but this must be an excessively uncommon event. The glands to which translation has occurred usually recover in a few days, without receiving any injury. The author has seen several cases in which the testes were injured—the damage consisting not in atrophy, but in an epididymitis, with occlusion of the spermatic duct.

Diagnosis.—The prevalence of an epidemic, the occurrence of swelling in the parotid gland with fever, and the subsidence of the swelling and fever in a few days, are clinical features which readily separate mumps from other affections. In children having bad teeth there may be produced a swelling of the parotid and submaxillary glands, but here the pain and swelling about the tooth will explain the nature of the case. Inflammation and suppuration of the parotid will be differentiated by the formation of pus and by the usual symptoms of glandular inflammation.

Treatment.—As this is a self-limited disease for which we have no remedy, it is wisest to attempt no perturbating treatment. Relief to the pain is best afforded by some warm applications, and by the internal use of morphine and quinine. A mild laxative should be administered, and, if the skin is hot and dry, the body may be sponged off with cold water, and some tincture of aconite administered. Recent observations have apparently demonstrated that pilocarpus possesses a peculiar curative power. This may be given in the form of the fluid extract, or of the alkaloid pilocarpine, and is well worthy of further trials. The patient should be kept in-doors, and every effort made to avoid the least contusion of the testes.

MALARIAL DISEASES.

INTERMITTENT AND REMITTENT FEVERS.

Definition.—*Malarial fevers* are characterized by their prevalence in certain regions of the world known to produce the poison, *malaria*, by their periodicity, and by the regular succession of the cold, hot, and sweating stage. Various designations have been applied to these forms of fever, such as *fever and ague*, *chills*, *bilious fever*, *bilious remittent*, etc.

Causes.—The great etiological factor is *malaria*. The telluric and other conditions favorable to the development of malaria exist largely in this country, along the Atlantic seaboard as far north as Boston; in all that great interior region drained by the Mississippi and its tributaries, the valley of the Sacramento on the Western coast, etc. For an exhaustive account, the reader is referred to the recent work of Lombard, or to Hirsch.* The presence in the atmosphere of a morbific principle, which is developed when certain atmospheric and telluric influences exist, is now almost universally admitted. Although the existence of such a principle is admitted, the attempts to isolate and define it have proved abortive, unless the recent discovery of Klebs and Tommasi-Crudeli supply the missing form.† The "Bacillus Malariæ," which they have discovered floating in the atmosphere of the Pontine marshes, produces paroxysms of intermittent fever in the animals subjected to its action by inoculation. If this discovery is confirmed, and these rod-like bodies are proved to be the cause of those phenomena which we call malarial fever, it will prove to be the first and most important step toward permanent eradication of the disease. Malaria is also called "marsh-miasm," because of the abundance of this poison about marshes. But not all marshes produce malaria. The "Dismal Swamp," for example, is free from marsh-miasm, although apparently well adapted to produce it. Its exemption is supposed to be due to the growth of the cypress-tree. Marshes, or moist alluvium, subject to annual overflow, and exposed to the action of the sun, by reason of evaporation or subsidence of the water, is peculiarly active in the production of the poison. Marshes that are partly brackish are worse than those entirely fresh. In this country malaria is produced more from the sandy alluvium of the river valleys subject to annual overflow and heated by the summer's sun. The alluvium and some very sandy soils of the malarial zone, not subject to overflow, also generate malaria, which is freed by turning up the soil. Cultivation and drainage, however, ultimately destroy the malaria-breeding grounds, and marshes, drained and planted, finally cease to produce the miasm. The malaria zone extends northwardly as far as the isothermal line of 59° to 59·8° Fahr., or to 63° north latitude.* It is the mean annual summer temperature, however, which determines the northern limits of malaria, and this pursues an irregular line which may be at some points above, at others below, the sixty-third parallel. One important factor is elevation, malaria not breeding above five thousand feet above the sea, which seems to be the maximum limit. The apparent exceptions to this afforded by the so-called "mountain fever" of Colorado will be alluded to hereafter. The period of the year during which malaria is most active is summer and fall—from June till November—for at this period only has the sun sufficient power. During the season of its greatest intensity, the poison may be carried up ravines to a considerable elevation, or to distant points. A position to the leeward of an infected locality is, therefore, particularly dangerous. That malaria is soluble in water and is contained in the surface-water of infected districts seems now to be well established. The author found the surface-water of Kansas to produce malarial fevers and cholera. Some trees possess the property of absorbing and fixing in their own structures noxious principles contained in the soil. The common sunflower, planted in moist lowlands, will render the air salubrious. The eucalyptus-tree has changed the nature of the malaria-breeding portions of Algiers, and is accomplishing the same sanitary result for the Campagna of Rome. The air is filtered of its disease-germs by passing through a belt of woodland ; even shrubbery a few feet high serves the same purpose, and protects those living to the leeward. All ages are susceptible to malarial poisoning ; and all races are equally so, except the black. Males are somewhat more liable, probably because they are more exposed to the causes. Women suffer more from the masked forms, as hemicrania, supra-orbital neuralgia, etc. All causes depressing the vital forces favor the reception of the poison and the outbreak of the disease. Especially is exposure to cold and dampness combined apt to cause an attack. Previous attacks increase the susceptibility. If those living in the midst of a malarious influence go from it into a region entirely free from all suspicion of the infection, an outbreak of the fever is apt to occur. When malarial infection is established in the system, all diseases occurring will have more or less of the periodical character. The form of the malarial disease occurring will depend on the condition of the system, and on the intensity of the poison itself.

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* For an account of the great interior valley of this continent, see the monumental work of Dr. Daniel Drake ("A Systematic Treatise, Historical, Etiological, and Practical, of the Principal Diseases of the Interior Valley of North America," page 723), for the reasons which induce him to accept the doctrine of the cryptogamic origin of malarial diseases.

† Klebs und Tommasi-Crudeli. "Studien über die Ursache des Wechselfiebers und über die Natur der Malaria," "Archiv für experimentelle Pathologie und Pharmacologie," Bd. xl, s. 311.

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* The forty-seventh parallel is given by Drake (*supra*) as the northern limit in this country, and the summer temperature of 60° Fahr.

Pathological Anatomy.—The changes caused by malarial poisoning are essentially the same, except degree, in all the forms in which the disease manifests itself, and two organs (the liver and spleen) are chiefly concerned. In acute cases, the spleen is much enlarged, splenic pulp greatly increased in relative quantity, and sometimes there are infarctions. Gangrene, abscess, and rupture of the spleen are accidents which have been observed in some cases of pernicious fever. In some chronic cases the spleen undergoes enormous enlargement; its texture is tough and smooth on section, and it has a grayish slate color This change consists in a hyperplasia of the trabeculæ with hypertrophy of the capsule, but in some cases the increased size of the organ is due to amyloid degeneration. When the organ attains to very large dimensions, it is known as "ague-cake." Usually, in chronic malarial poisoning, the spleen is somewhat enlarged, but not so much increased as to be called ague-cake, the change consisting in a diminution of the splenic pulp and an hypertrophy of the trabeculæ and capsule. The color of the spleen is grayish or slate, due to pigment deposits, which are found in greatest abundance in the walls of the blood-vessels, where it is deposited by disintegration of the red globules. Important changes take place in the liver. During an intermittent the liver becomes hyperæmic and swollen, and, if jaundice is present, very much enlarged, stained with pigment, and the portal capillaries distended with blood, and the gall-bladder filled with thick, tarry, dark-brown bile. In chronic cases the liver has a grayish tint, due to pigment deposits along the vessels; it is firm in texture, and the divided parts preserve sharp outlines; the hepatic cells are pale and filled with fat-granules. The intestinal canal also presents characteristic changes. During an acute attack there are extensive and considerable hyperæmia of the mucous membrane and more or less thickening and elevation of the solitary and agminated glands. In the chronic cases the intestinal mucous membrane has a general slate-colored hue, due to pigmentation of the capillaries. The glands, solitary and agminated, are thickened and enlarged from accumulation of their contents and hyperæmia, and thickly disseminated through the groups of Peyer are the black orifices of the follicles of Lieberkuhn. The kidneys are also affected by characteristic changes: hyperæmia during the acute attack, and subsequent alterations, as thickening of the basement membrane, the tubules filled with cast-off epithelium, the interstitial connective tissue proliferating, and more or less amyloid change in the Malpighian tufts and small arteries.* The brain and spinal cord do not escape. In ordinary cases during an acute attack, there is more or less hyperæmia of the brain; in pernicious remittent, capillary pigment embolisms and minute extravasations occur; but more usually the condition is that of hyperæmia and œdema of the membranes and of the cerebral matter. In the lungs there may be infarctions, croupous pneumonia, etc. The heart is flabby, its muscular fiber easily torn, the right cavities distended with soft, black coagula, very loose. The changes in the blood have not been studied with accuracy. Bence Jones's discovery of a fluorescent substance in the blood and tissues has not thrown any light on the question, since this substance or rather reaction is very widely distributed and is without importance. It is true, Pepper and Rhoads found this substance diminished by malarial fever, but nothing has resulted from these observations. The white corpuscles are much increased in numbers relatively, but the most important change in the composition of the blood is the formation of pigment from the hæmoglobulin, the hæmatin is set free, and is found in all the principal organs associated with the vessel-walls, and rarely collected in masses, and forming capillary embolisms.

Symptoms.—*Prodromal Stage.*—A certain period elapses after exposure before there is any disturbance in the functions. This period of incubation varies from a few hours to many weeks, the variations being due to the intensity of the poison and the susceptibility of the individual. The average which is most usual is fourteen days. In a large proportion of cases there are symptoms indicating that the infection is working. These are called prodromes. The patient has a feeling of lassitude and weariness; he suffers with backache and general muscular soreness; he has an irresistible inclination to yawn and stretch, especially in the early morning, and on cold, damp days; his head aches, tongue is coated, stomach is squeamish; toward evening his skin becomes warm and dry, his sleep is disturbed by dreams, and in the early morning a profuse sweat occurs. In other cases the prodromes consist merely in a coated tongue, yellow sclerotic, and a general yellowish hue of the skin, languor, loss of appetite, and constipation; the urine is loaded with bile-pigment, and deposits an abundance of urates. Gradually thus may the patient drift into a paroxysm of fever, without there being any distinct initial symptom—the form assumed developing by a process of selection, as it were, out of the material offered. Or the disease may begin abruptly in the midst of apparently full health, or during the puerperal state, or in the course of chronic malarial poisoning.

Intermittent Fever.—*Ague and Fever.*—There are three distinct events in every paroxysm of intermittent fever : the chill, the fever, and the sweat. When the chill comes on, there is a feeling of wretchedness, of weariness, and illness. There occur headache, backache, and soreness in the muscles of the extremities. Creeping chills are felt along the back, there are gaping and præcordial oppression, the whole surface grows cold, and, feeling extremely weary and depressed, the patient gladly betakes himself to bed ; but the coldness intensifies, no matter how much covering is piled on ; the fingers become blue, the lips blue, the nose pinched, the countenance shrunken, and the chilliness is now aggravated into shuddering. One fit after another of shuddering comes on ; the teeth rattle together ; the bed shakes. Meanwhile the pains in the head and back and limbs continue ; there is extreme thirst, and often nausea and vomiting ; respiration is quick and sighing, the voice is weak and tremulous ; the pulse is small, rapid, and the tension high ; the urine is pale, watery, and increased in quantity. Notwithstanding the overpowering sense of coldness, it is found to be objective, for the temperature begins to rise with the onset of the chill, the thermometer indicating fever whether in the axilla, mouth, or rectum. The duration of the chill varies from a mere instantaneous chilliness to several hours of shaking, the usual length of the ague being a quarter to a half hour. The chill does not terminate abruptly. The shaking subsides slowly, as a feeling of warmth gradually diffuses outwardly, or flashes occasionally through the limbs. After a time the body feels hot, the extremities grow warm, the pulse becomes fuller and stronger, the blueness of the skin is replaced by a red blush, the face is full instead of retracted, flushed instead of pallid. The pains in the back and limbs disappear, but the headache rather increases, and throbbing is felt in the temples, and with each pulsation of the carotid. The pulse grows full, rapid, and strong; respiration is more frequent and easy. The head becomes hot, feels full ; there are noises in the ears ; vertigo and nausea are experienced on the attempt to get up ; the ideas are confused, and the mind is dull, and there may be excitement and delirium. The usual symptoms attend this feverish state—there are thirst, a dry mouth, constipation, high-colored, scanty, and acid urine. The duration of this stage varies from an hour or two to ten or twelve, and it is succeeded by the third or *sweating stage*. While the fever is raging, a gentle moisture appears on the forehead and face, and more abundantly in the axilla, groin, between the thighs, and then on the skin. Presently the moisture increases to drops, and finally pours off, wetting the shirt and the sheets. As the sweating progresses, the fever declines, the pulse becomes softer and its tension is lowered ; the headache and other pains and the general muscular soreness cease ; the mouth gets moist and the thirst lessens ; the respiration becomes easy and regular, and the patient, although exhausted, experiences a feeling of comfort and well-being, and often falls asleep. The sweat is acid in reaction, is rich in salts, and contains a large quantity of organic matter with fat acids, to which its animal odor is chiefly due. The urine also is acid, has a high color owing to a quantity of pigment, and contains much uric acid and urates, which are deposited abundantly on cooling. The amount of urea discharged corresponds closely with the range of temperature, and, as soon as the fit of ague begins, the production of urea increases (Ringer). A sudden decline in the amount of urea takes place during the sweating stage, and in the apyretic interval it is below the normal.* The excretion of chloride of sodium also is always increased greatly during the cold and hot stage of an ague paroxysm. These facts indicate that the increased temperature of the febrile movement represents the consumption of tissue. When the paroxysm is entirely ended by the completion of the sweating stage, in about twelve hours, on the average, from the beginning of the seizure, the patient presents evidences of the revolution through which he has passed. There is experienced a sense of exhaustion, and the functions generally are depressed; the tongue coated, the appetite poor, the epigastrium and hypochondriac regions more or less uneasy and sensitive to pressure, and the skin is slightly or considerably jaundiced. Not every ague attack is so severe, and great variations are observed as regards the several stages. Thus the chill may be a mere creeping or crawling sense of coolness along the spine, while the fever and sweat may be extremely severe. Again, the chill may be pronounced and the fever and sweat trivial; or there may be profuse sweating at regular intervals, without any but the most trivial and transient disturbances in other respects.

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* The author gives the results of numerous observations and studies made during his service in the regular army, from 1857 to 1864. (See his contributions to United States "Sanitary Commission Memoirs," medical volume.)

Course, Duration, and Termination.—After a certain interval, which is different in the several types of fever, the paroxysm recurs, and there are again presented the phenomena of chill, fever, and sweat. Intermittent fever follows a definite law of periodicity. Sometimes the paroxysms occur daily, coming on at a special time with nearly uniform particularity. This variety or type is known as *quotidian intermittent.* Again, the paroxysms occur on alternate days—on the third day, including the days of attack—and are hence known as *tertian intermittent.* In the temperate malarious regions the tertian form is the most frequent. There is still a third variety, in which the paroxysms occur on the fourth day, including the days of illness, and hence is known as *quartan intermittent.* This last variety is uncommon. Sometimes two distinct paroxysms occur on the same day, and hence we have *double quotidian*, *double tertian*, etc. The author has encountered two cases of double quotidian in the puerperal state. Other eccentricities have been observed. Thus, a quotidian may have on alternate days corresponding paroxysms as to time and character, and may consist of two tertians. Such a variation is sometimes called a double tertian. The *triple tertian* is a variety in which there are two distinct paroxysms on one day and one paroxysm on the next; the *duplicated tertian* has two paroxysms on alternate days; and, finally, the *double quartan* has a paroxysm on one day, a milder one the next day, and a day without fever. The duration of a paroxysm of fever varies with the type: the quotidian lasts from eight to twelve hours, the tertian from six to eight, and the quartan from four to six. The paroxysms do not always occur at the same hour; if uninterfered with they anticipate, the second occurring a little earlier than the first, and the third earlier than the second. On the other hand, as the force of the attack is declining, the paroxysms are postponed. The quotidian usually begin in the early morning, the tertian toward or at noon; if not interfered with by treatment, an intermittent will ultimately terminate spontaneously, but the period at which this result will be reached depends on the climate, constitution, season, degree, in which the system has been poisoned by malaria, etc. Very mild quotidians may terminate in a month, tertians in two months or longer, and quartans many months. When malarial poisoning has thoroughly occurred, the disposition to attacks continues for a long period—often for years. Exposure to cold, errors of diet, fatigue, mental anxiety—a variety of causes, of sufficient force to disturb the functions—may excite a new attack. Very often a change of type ensues: the quotidian may become a tertian, or the gravity of the case is increased—a remittent succeeding to an intermittent fever. It is rare for an intermittent fever to terminate in death directly, but indirectly, through the various alterations occurring in malarial poisoning, a large mortality results. The course of intermittent is much diversified by the variations from the typical form known as *masked intermittent.* When an attack has been interrupted by the exhibition of the usual remedies, there may occur at the regular periods subsequently a mere temporary rise of temperature, a profuse sweat, a copious urinary discharge, an attack of diarrhœa, etc. With or without any previous manifestation of fever, those affected with malaria may suffer with various substitution diseases, as intermittent hæmaturia, pulmonary hæmorrhage, bronchitis, coryza, iritis, jaundice, diarrhœa or dysentery, vomiting, urticaria, roseola, and numerous other maladies. These substitution diseases agree in coming on at a fixed hour or nearly so, in disappearing after a time without any apparent reason, in coming on again at the appointed time or anticipating a little, and in yielding promptly to the anti-periodic while obstinately resisting other means of treatment Probably the most common of these substitution diseases is *neuralgia* and the most usual position of this, the ophthalmic division of the fifth; but it may occur in the other divisions of this nerve—in the occipital nerve, in the sciatic, and elsewhere. In what position soever the neuralgia appears, the attacks are periodical, and usually quotidian. When it occurs in the ophthalmic division, there are intense pain in the region of the eye and forehead and throbbing temples, the conjunctiva is injected, and the eyelids are swollen; general *malaise*, nausea and vomiting, some chilliness, elevation of temperature, and sweating are the systemic symptoms, which associate these cases with the ordinary intermittents. When sciatica occurs it may assume the intermittent or remittent form, is on the right side in the majority, and is sometimes accompanied by clonic spasms. Not frequently, attacks occur in the cardiac nerves, producing the phenomena of angina pectoris, viz., præcordial oppression and pain, a sense of impending death, great difficulty of breathing, a slow, hard pulse, cold skin, blue lips and fingers, ending with free eructations of gas, the discharge of a quantity of pale, watery urine, etc. Various nervous diseases, as delirium, puerperal mania, hallucinations, coma vigil, etc., have occurred, as those above mentioned, in substitution of malarial attacks. Besides the intermissions, the regularity in the periods of recurrence, and the promptness with which they yield to quinine, these substitution maladies may be accompanied by some of the other objective phenomena of malarial fever.

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* Dr. Joseph Jones, "Trans. Amer. Med. Association," 1859, vol. xii, p. 507; Sydney Ringer, "Medico-Chirurg. Trans.," second series, 1859, vol. xxiv, p. 361; Dr. Parkes, "On the Composition of the Urine in Health and in Disease," London, 1860, p. 235.

Pernicious Intermittent.—In those parts of the United States where the malaria is most concentrated and the malarial fevers most severe, the ordinary intermittent may assume a most formidable character, termed *pernicious* in scientific works, and popularly known as *congestive*. That an attack of intermittent will assume a pernicious character is not announced in advance. Sometimes the condition of exhaustion induced by a severe attack of cholera morbus may invite a paroxysm which assumes the pernicious character, or the state of the patient may be rendered unfavorable by some other malady, or there may be present some symptoms of cerebral disturbance, but in general there is nothing to indicate the approach of the severe type. Usually, the case has the ordinary aspect of an intermittent for the first, second, and third paroxysm. There may be a gradual increase in the severity of each attack, or the usual type may be followed by a pernicious one. It is not often that the first pernicious attack proves fatal, but a repetition of them becomes more and more dangerous, and after the first any succeeding attack may be fatal. The pernicious attacks assume several forms—the algid, choleriform, diaphoretic, the pneumonic, the nephritic, and the cerebro-spinal.* In the *algid* form the depression of the heart, which is its distinctive feature, comes on either in the fever or sweating stage. While intense internal heat is experienced by the patient, the surface becomes cold, livid, and cyanosed, the pulse small and exceedingly rapid, the action of the heart feeble ; the skin is covered with a cold, sticky sweat, but the mind is undisturbed. If death occurs, the condition of coldness and depression increases, but if recovery, after a longer or shorter duration of the algid state, the action of the heart grows a little stronger, and gradually warmth is restored to the surface. In the *choleraic* variety of pernicious fever there is produced an algid state resembling that of cholera, by an uncontrollable vomiting and purging, and the resemblance is carried to the stage of reaction ; for if the patient emerge from the condition of collapse he experiences the fever of reaction—the typhoid state—which occurs under similar circumstances in cholera. In the *sweating* or *diaphoretic* variety of pernicious intermittent no notable change in the demeanor of the case takes place until the stage of sweating arrives, when, not only does an enormous transpiration occur through the skin, but the temperature falls below the normal, the circulation becomes exceedingly depressed, the surface cold and cyanosed ; the urinary secretion is greatly diminished or totally suppressed, and in many cases there are passed large, whitish stools, without bile. Under such circumstances there may be more or less jaundice, and by many authors those cases characterized by a marked biliary derangement are erected into a distinct class, as *pernicious icteric* (Jaccoud). When the vaso-motor disturbance, which underlies the forms of pernicious intermittent, already described, is precipitated on some internal organ, there will ensue, in addition to the condition of coldness, cyanosis, and feeble circulation, the symptoms of some particular internal malady—pneumonia or pleurisy, for example. A malarial pneumonia pursuing the ordinary course, the symptoms remitting in accordance with the type of the malarial fever, will, if the pernicious symptoms set in, assume, in a short time, a condition of extreme danger, owing to the disturbance in the pulmonary circulation. When the vaso-motor derangement affects the kidneys during the course of pernicious intermittent, there is produced the *nephritic form* of pernicious fever, and the signs are hæmaturia, albuminuria, or suppression of urine. The most common form of pernicious intermittent is that affecting the nervous centers. There are usually some preliminary symptoms, as headache, vertigo, and a soporose state, which are present during the first paroxysms, or in the interval preceding the pernicious attack. During the fever stage the patient falls into a profound coma, and this is all the more dangerous, because it may resemble natural sleep. In the first attack, the patient usually rallies during the sweating stage, in twelve to twenty-four hours, or the coma may simply deepen, the heart become more and more depressed until death. The succeeding attacks are usually fatal. This comatose form may assume an appearance of apparent death, the patient being in a cataleptic condition, or it may be preceded by fainting-fits, a state of genuine coma then coming on. In still other cases this cerebral form of pernicious fever may assume the appearance of maniacal delirium, or it may affect the brain and cord simultaneously, causing tonic and clonic spasms, etc. In this country the most frequent varieties of pernicious intermittent are the algid, the choleraic, the pneumonic, and the comatose.

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* Jaccoud, *op. cit.*, p. 605.

Sequelæ of Intermittent Fever.—When attacks of intermittent fever have been interrupted by appropriate treatment, relapses are apt to occur. In fact, by the treatment only the objective phenomena of fever may have been removed, and consequently but a certain time will be required to develop new paroxysms. In cases thus temporarily suspended and apparently well, it will be found on close inspection that there are still occurring in regular sequence certain disturbances. The thermometer may show some slight elevation of temperature; there may be a distinct sweat, or a profuse urinary discharge may occur, and, after a period determined by the type, the paroxysms will recur. These relapses are said to appear on the seventh, fourteenth, and twenty-first days, but it is more correct to state that the periods of recurrence are multiples of the first or former attacks. If, for example, the case is tertian, the first relapse would occur on the sixth day ; if quotidian, relapses would take place on the third, sixth, ninth, and twelfth days ; and thus on. Not only the regular cases, but the various masked and pernicious forms, manifest the same tendency and pursue the same laws as regards the relapses. The tendency to the occurrence of relapses is much affected by age—is much greater under twenty, and declines rapidly after twenty. The time at which they occur varies greatly, from one week to six months, but the probability of a relapse is very slight after six weeks have passed. The type of the disease frequently changes in undergoing a relapse, the tendency being to more frequent attacks, the tertians becoming quotidian. The tendency to relapses is due to the persistence of the conditions which determined the first seizure. The result of the long-continued action of malaria is most disastrous. The blood loses its red globules, while the white diminish in size and increase in number ; the ankles become œdematous ; the liver and spleen enlarge ; the skin is yellow, earthy, or jaundiced ; the body emaciates ; the appetite is poor, the digestion feeble, the stools clay-colored, and the urine may contain albumen, and is deeply colored with bile-pigment ; fluid accumulates in the peritoneal cavity, etc. Palpitation of the heart and a venous hum over the course of the great vessels occur because of the watery state of the blood, and for the same reason epistaxis takes place and the menses become profuse. The changes which affect the composition of the blood are due to various causes—to the interference by stomach and intestinal troubles with the primary assimilation, to the morbid state of the blood-making organs, especially to the destruction in the spleen of the red-blood globules, and to the conversion of hæmatin into pigment, which we have shown to take place at various points. An important fact is the accumulation of this pigment, and its almost universal distribution throughout the body. The mischief done by pigment embolisms is doubtless very great. Besides those changes belonging to chronic malarial intoxication and the sequelæ above mentioned, there are various maladies of high importance, which may have their origin in the malarial cachexia. Among these are nephritis, amyloid degeneration of the liver, kidneys, spleen, and intestinal glands; sclerosis of the liver, anæmia, dropsy, tuberculosis, neuralgia, epilepsy, hemiplegia, mania with suicidal tendency, etc.

Diagnosis.—A case of intermittent, complete at all points, could hardly be confounded with any other malady. It may be mistaken for pyæmia, in which there are chills, fever, and sweats, with an apyretic interval. It differs, however, from pyæmia in its origin, and in the clinical course; intermittent is due to a supposed miasm—pyæmia to wounds, suppuration of veins, etc.; intermittent is regular in its course—pyæmia is very irregular, no defined intervals occurring; intermittent is a benign affection, promptly cured by quinia—pyæmia is a fatal disease, over which quinia has no influence. Masked intermittents are differentiated from the local maladies whose form they assume, by the fact that malaria is abundant, that these diseases are distinctly periodical, and that they yield to the remedies for malarial diseases. The diagnosis of the various pernicious forms is very difficult. It ought to be remembered that the pernicious attack has occurred at a time when the regular paroxysm is due, and that probably a strong malarial influence prevails. The comatose variety is often preceded by symptoms indicative of the disturbance in the intracranial circulation, such as headache, vertigo, sopor, etc.

Remittent Fever.—The remittent fever of this country is known as *bilious fever* and *bilious remittent fever*. The designation *bilious* has been applied because of the prominence of the symptoms referable to the hepatic function. Every summer and fall this disease prevails largely through the South and West. The author saw in Kansas, in 1857, at the military post of Fort Leavenworth, a great many examples of the severe form of remittent fever prevalent in that locality. The cases of remittent are divisible into three groups—*mild*, *severe*, and *grave*. These divisions, generally recognized by systematic writers, are based on clinical experience. In the mildest form the fever continues for four or five days, when distinct intermissions occur; the remissions are well defined from the beginning, and increase day by day into the complete intermission. Usually an attack of remittent fever is preceded or accompanied by a coated tongue, yellow and thick; a heavy, offensive breath; nausea and vomiting—the matters ejected consisting, for the most part, of acid mucus and bile; violent headache, especially of the frontal region, ringing in the ears, throbbing temples, and a chill of moderate severity, which marks the real onset of the disease. The remission is every day (quotidian type), or on alternate days (tertian type), and is marked by a distinct sweat, which coincides with the decline of temperature. More or less chilliness, sometimes a well-defined chill, begins the new paroxysm. Restlessness and wakefulness at night, bleeding at the nose, a slight bronchitis, and an eruption of herpes, are also symptoms of this form. In the *severe form* the fever is less broken by remissions, and assumes a type approaching the continued. About the third day there are beginning symptoms of cerebral derangement, as stupor and delirium; the tongue is dry and cracked; the spleen and liver are enlarged and swollen; a well-marked icterus stains the skin, and in some cases pernicious symptoms are developed out of a complicating dysentery or pneumonia. Such a case may extend over two weeks, and gradually abate into an intermittent, or terminate fatally, with pernicious phenomena, in collapse. In the *grave form* the case may begin as in the severe variety; in the first week the exacerbations and remissions will be irregular, perhaps, with a tendency, constantly increasing, toward a continued type, delirium and stupor coming on, and deepening into coma. Instead of a gradual progress toward a typhoid state, the case may begin with serious symptoms, and in a few hours delirium, jaundice, hæmorrhages, albuminuria, or suppression of urine may appear. In other cases, choleraic symptoms or dysentery may come on, purulent effusions into the serous sacs may occur, a pneumonia may develop, abscess may form in the liver, and gangrene of the skin may result. A form of remittent fever of great severity, and having close analogies with yellow fever, is that known as the *hæmorrhagic bilious fever*. It may commence as an ordinary intermittent, but the grave symptoms rapidly develop. The chills are protracted and violent, intense headache and backache are then experienced, a burning pain passes from the pharynx to the stomach, very depressing nausea now comes on with vomiting of bilious matter, obstinate constipation is succeeded by a bilious diarrhœa, the urine is copious and dark in color, the skin assumes an icteric hue, and very considerable swelling of the spleen and liver occurs. Meanwhile the fever becomes remittent and the remissions less and less marked, the pulse rapidly declines in volume and strength, the skin is covered with a cold sweat, the features shrink, hæmorrhages occur from the mucous surfaces, the urine lessens greatly in quantity or is entirely suppressed, and the fatal result is reached in an increasing coma. Notwithstanding the formidable character of this variety of remittent fever, a fatal result is not inevitable, if the subject be vigorous, and the treatment properly carried out before the onset of coma, which may appear on the fourth, fifth, or sixth day. So strong is the resemblance of these cases to yellow fever that they are doubtless often confounded during the epidemic prevalence of the latter. No means of distinction between them is so satisfactory as the action of quinine, which will arrest the one but not affect the other.

Treatment.—The questions of public and private hygiene involved in the prevention of malaria are beyond the scope of this work. The direction which the investigation of physicians should take is indicated in the etiological chapter. The measures of prophylaxis, as affecting individuals, must, however, receive some attention. Those living in malarious regions, susceptible to the action of the poison, must avoid all excesses of every kind, exposure to fatigue, to heat, and to rapid alternations of temperature. Exposure to the night air and to the early morning air is also to be avoided. Before leaving the house in the morning a substantial breakfast should be taken, and a prophylactic dose of quinine, if the season of malarial production has arrived—summer and fall. The experience now accumulated as to the prophylactic power of quinine puts this question beyond controversy. The English naval experience on the coast of Africa, the military experiences in India and Africa, and our own experience during the civil war, have demonstrated that the daily administration of a sufficient dose will procure immunity against malarial infection. The quantity required for this purpose is differently stated, but should be determined by the supposed intensity of the malarial poison, and may be put at from five to ten grains daily. It is best administered in the early morning, and in some black coffee, or dissolved by the aid of sulphuric acid in water, in pill form, or simply in water. The practice pursued in our army during the war, of giving quinine in whisky, is wrong in principle, and the results were not good, therapeutically or morally. The effects of quinine as a prophylactic are much more certain than when used in a corresponding way to prevent relapses. In fact, it is much easier to prevent than to cure the disease. If there is no time to prevent the paroxysm, we possess means to abort it at the chill stage. The expedients resorted to for this purpose are very numerous, and include nitrite-of-amyl inhalations ; chloroform by inhalation and by the stomach ; the hypodermatic injection of morphine and of pilocarpine. From a half-drachm to a drachm (fluid) of chloroform, given in some sweetened water, by the stomach, or administered by inhalation, will usually arrest the chill, and greatly lessen the severity and duration of the succeeding stages. Amyl nitrite is also quite efficient in bringing on reaction and abbreviating the chill stage, but it exercises little or no influence on the other stages. Recent observations seem to prove that pilocarpine, of all the remedies hitherto proposed for this purpose, exercises the most remarkable influence.* If administered as the chill is coming on, it stops it, and substitutes a sweating stage, thus preventing the full evolution of the paroxysm. The most remarkable point is that the disease seems arrested, and relapses prevented, in a considerable proportion of the cases. If these observations are confirmed, we shall have in pilocarpine the most useful remedy in the treatment of intermittents. From one twelfth to one sixth grain of the nitrate or muriate of pilocarpine, given hypodermatically, is the appropriate dose for an adult, and this should be given as the chill is about to occur. A corresponding dose (one sixth to one fourth grain) can be given by the stomach half an hour before the chill-time. If the chill has anything of the pernicious character about it, the most efficient remedy is the hypodermatic injection of morphine and atropine, or of morphine alone. In any of the modes in which the pernicious attacks come on, the remedies are two—morphine and quinine—and the mode of administration subcutaneous. The usual means of applying artificial heat are of course to be used, but no time should be expended on anything until morphine and quinine shall have been injected subcutaneously. From one twelfth to one fourth of a grain of morphine can be given to an adult. Maximum doses of quinine are required. Much difficulty has hitherto been experienced in preparing a suitable solution of quinine. As the muriate of quinine and the bromide are soluble to a much larger extent than the sulphate, they may be used for solution in water only; but as the quantity required is so great, a solution of the sulphate, dissolved by the aid of sulphuric acid, is generally preferred.* The dose of quinine injected in a pernicious case should not be less than twenty grains, and this may be repeated two or three times until reaction is established. In the absence of the method or means of hypodermatic injection, quinine and morphine may be administered by the rectum, if insensibility or irritability of the stomach prevents the introduction of remedies into that viscus. If the approach of a pernicious intermittent is indicated by the presence of head-symptoms—drowsiness, headache, vertigo, etc.—the administration of full doses of quinine should not be delayed.

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* Dr. Griswold, August 16, 1879, "New York Medical Record."

In the treatment of ordinary intermittents, our attention is directed to the prevention of future attacks. Although no preparatory treatment is actually required, better results are obtained if the gastro-intestinal derangement is removed. If the tongue is heavily furred, the stomach irritable, and the bowels constipated, the absorption of quinine is much hindered and its powers lessened. A grain of calomel, followed in four or six hours by a Sedlitz-powder, or the latter without the calomel, will assist in the absorption of the quinine. The old plan of an emetic, followed by "ten of ten"—ten of calomel, ten of jalap—is no longer pursued. Opinions still differ as to the period of administration, and the dose of quinine, in the treatment of intermittent fever; but these differences exist among those only who have but limited experience in the management of severe intermittents. The question is, shall we use small doses, frequently repeated in the interval, or a single full dose at the proper period before the access of the paroxysm? The latter is better, for these reasons: the whole effect of the quinine is obtained at the right time, a less quantity suffices, and the curative effect is greater. As the elimination of quinine takes place with considerable rapidity, appearing in the urine in three hours after it is swallowed, it is obvious that, if the administration has been distributed over twelve hours, the effects of the first doses are expended before the last are given. The amount necessary to arrest the paroxysms should, therefore, be given at a dose, or within a short period, and at a time preceding the chill sufficient to obtain the maximum effect, which is about three hours. For an ordinary intermittent from fifteen to twenty grains of quinine are necessary to stop the paroxysms. To prevent relapses, quinine must be given at certain periods: on the second or third day, and on the fourth and sixth days after the date of the first administration, according to the type. Having in view the tendency to relapse at subsequent periods, quinine should be again given on the twelfth to the fourteenth, and on the nineteenth to the twenty-first days. As, in cases of malarial cachexia, we have to deal with certain morbid conditions of the liver, spleen, intestines, blood, etc., attention must be given to them if we would effect a cure. To improve the condition of the blood, the chalybeates, notably the sulphate of iron, must be employed; and these remedies are the more efficacious if combined with arsenic and other tonics. During the intervals between the administration of quinine, the remedies best adapted to the existing state of malarial cachexia are, besides iron, arsenic and eucalyptus. Various substitutes for the expensive quinine are now largely administered. Probably the best of them are the combined alkaloids of cinchona in an impure form, as used by the authorities of India. Quinidine may be prescribed in the same quantity as quinine, and seems about as effective. Cinchonine is also quite effective in twice the quantity as quinine. The author has found the salicylate of cinchonidine quite a good antiperiodic, and next, probably, to the salts of quinine in power. Salicylic acid has some antiperiodic property, but greatly inferior to quinine; it has been combined with quinine to form salicylate, but its precise value has not been shown. Eucalyptus is a most useful antiperiodic, but it is adapted rather to the treatment of malarial cachexia, and to prevent relapses. Iodine possesses a high degree of utility in the treatment of malarial intermittents, and may be used in substitution for quinine, or to remove some of the secondary lesions. Lugol's solution is a convenient form in which to administer it. The combination of iodine and carbolic acid is highly efficient (℞ Acid. carbol. ʒ j, tinct. iodi comp. ʒ iij. M. Sig. Four drops every four hours in sufficient water). This combination may be depended on exclusively in some cases. For the removal of the various morbid alterations caused by malaria, the combination of iodide of ammonium and arsenic is most effective (to a solution of iodide of ammonium, giving five grains to the dose, add three drops of Fowler's solution). The practitioner will find this most useful in cases of chronic malarial poisoning with frequent intermittents. For the treatment of enlarged spleen there is, besides the exhibition of quinine, no remedy more efficacious than the ointment of the red iodide of mercury, which is rubbed in daily over the splenic region in the sunshine, until soreness of the skin compels a suspension. For the gastro-intestinal catarrh, the duodenal catarrh, and the catarrhal jaundice, which occur so frequently in malarious regions, with or without any febrile movement, the most serviceable remedies are two, the phosphate or benzoate of soda, three times a day, and a morning and evening dose of ten grains of quinine.

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* ℞ Quininæ disulph., gr. 50; acid. sulphuric. dil., ♏ 100; aquæ font., ℥j; acid. carbol. liq., ♏ 5. Solve.

For various formulæ, see "Manual of Hypodermic Medication," by the author of this work, third edition, p. 213.

NOTE.—The carbamidated muriate of quinine—a combination of muriate of quinine and urea—has been found to be the most effective as it is the most soluble of all the preparations, and is preferable to all others.

In *the treatment of remittent fever* the same general plan is to be pursued as in the management of intermittents. It is not necessary to await the remission, but the antiperiodic may be given at once, yet it is certainly true that the remedy in corresponding dose is much more efficient if given during the sweating. The author's first experience in the administration of large doses of quinine was gained under that able physician and medical officer, the late surgeon John M. Cuyler, M. D., of the Army Medical Staff, then stationed (1857) at Fort Leavenworth, Kansas. The author, a recent graduate in medicine, and just then admitted to the army, was very fortunate in being able to witness the practice of so experienced and able a physician. The large hospital of the post contained a number of the severe remittent fevers of that locality. They were broken up into intermittents and sent out of the hospital in a week, usually by the routine prescription of thirty grains of quinine the first morning, twenty the second, fifteen the third, and ten the fourth—single doses, and all taken at once. As remittent fever is due to a more intense and concentrated poison, no delay in the efficient use of quinine is proper; otherwise, it may lapse into the typhoid state, and be confounded with typhoid fever. The intermittent remainder requires the same management as an ordinary intermittent. Should there be, as is usual, great irritability of the stomach, quinine solution can be given by the rectum, and the usual remedies applied for the relief of the nausea and vomiting. If the rectum is also irritable and rejects the remedy, it must then be given hypodermatically. Whenever it is practicable to do so, the antiperiodic should be administered during the remission in the sweating stage. The almost numberless masked intermittents and remittents require the same management as an ordinary case of intermittent, except that they are more difficult to arrest and require maximum doses of quinine.

DISORDERS OF NUTRITION.

SCROFULA.

Definition.—By *scrofula* is meant a constitutional dyscrasia, hereditary or acquired, characterized by changes inflammatory and hyperplastic, occurring for the most part in the lymphatic system, the skin, mucous membranes, connective tissue, osseous structures, and viscera. Scrofula is also known as *struma*, the *strumous diathesis*, *tuberculosis*, the *tuberculous diathesis*, etc.

Causes.—Heredity is the most influential factor in its pathogenesis, but it is the predisposition and not the disease itself which is inherited. Those cases are said to be *innate* in which, owing to conditions present in the parents, not themselves strumous, a scrofulous constitution is transmitted to their offspring. Such conditions are old age, blood-relations, cachexia of syphilis, etc., which existing in the parents, the offspring may possess the strumous constitution. Acquired scrofula is the product of various evil hygienic influences, as crowding, bad air, poor food, insufficient clothing, overwork, especially in youth, and in dark, damp, and crowded apartments. Recent observations, especially those of Cohnheim, which indicate the essentially infective nature of tubercle—a product of scrofula—show the great danger of inducing tuberculosis in children by the consumption of milk from tuberculous cows. It is probable that many cases of acquired scrofula, especially in cities, are derived from this source. If a scrofulous predisposition exist in a latent state, it may be roused into activity by various causes. Certain diseases, as measles, whooping-cough, typhoid fever, etc., will have this effect. Scrofula manifests itself usually about the time of the first dentition, and increases from the third to the seventh year. It is rare for the manifestations to appear only after puberty. Glandular affections do not often occur before the second year. Scrofula prevails under all conditions of soil, climate, and elevation, but it occurs most frequently in those countries where crowding, bad air, and the other hygienic evils of dense populations are most abundant.

Pathological Anatomy.—The anatomical changes occur in the lymphatics, the skin, the mucous membranes, the bones and the viscera. As regards the lymphatics, the cervical, bronchial, mesenteric, inguinal, and others are affected by two processes—one, and the simplest, consisting of hyperplasia of the gland-elements ; the other, and more complex, being the formation and subsequent caseation of tubercle. From the hyperphasia may proceed an inflammatory process, involving not only the gland but the adjacent connective tissue and skin ; suppuration takes place, abscesses form, and fistulous tracks and sinuses are made by the discharge of pus. The first step in the caseation of the gland is an enlargement by hyperplasia, then miliary tubercles form, or, without them, cheesy masses develop in distinct layers from the hyperplastic materials, and ultimately the whole gland becomes caseous. It is a disputed question whether there is a necessary development of the miliary tubercle precedent to cheesy degeneration, or whether the process of caseation develops out of the new hyperplastic materials. It is probable, as stated above, that both processes share in the production of the result. The cutaneous manifestations of scrofula consist in eczematous and impetiginous eruptions, situated on the face, scalp, or behind the ears ; and at the nose prominent pustules of impetigo with thick yellow crusts and suppurating beneath, the adjacent nasal mucous membrane ulcerating, are the characteristic appearances. The mucous manifestations of scrofula are usually situated at or near the junction of the membrane with the external integument, and the cutaneous lesions are associated with the mucous. Thus, impetigo of the lip is coincident with a scrofulous coryza ; otitis externa with retro-auricular eczema ; catarrhal conjunctivitis with eczema of the neighboring cheek. Strumous coryza after some years becomes an ozœna, and affects by contiguity the post-nasal fossa. The mucous membrane of the larynx and bronchi, of the genito-urinary tract, and of the intestinal canal, may also be attacked. The broncho-pulmonary membrane is a favorite seat of strumous changes, and here they manifest a strong tendency to ulcerative action. The connective tissue is affected by abscesses ; the joints become the seat of chronic synovial disease, of erosions, caries, etc. ; the periosteum inflames, the bones also, and caries and necrosis are ultimate results of the changes, or the primary disease may arise in the spongy portion of bone, especially in the vertebra, and the epiphyses of the long bones. In the viscera the most important of the lesions due to scrofula are those of the lungs—cheesy pneumonia, phthisis, etc., and those of the cerebellum, producing large, cheesy nodules. Amyloid degeneration of the liver, spleen, and kidney; caseous infiltration of the supra-renal capsules and tuberculosis of the testes are also products of the strumous diathesis.

Symptoms.—There are two distinct types of the scrofulous constitution, the light and the dark, the *irritative* and the *torpid*. In the former the skin is white and transparent, the veins showing through with great distinctness, and blushing taking place with extreme facility; the hair is soft, long, and fine in texture, and usually of light shade; the eyes are large, blue, and brilliant, the pupils dilated, the sclerotic pearly; the muscles are soft and flabby, the weight in proportion to size small; the mental development is precocious, and puberty anticipates the usual period.* The torpid form is characterized by a thick, coarse, and rather dark skin, a considerable preponderance of adipose tissue, the muscles being weak and relaxed; the body is gross, the appearance puffy, the habit torpid and heavy; the head is relatively large, the nose short and stubby, the upper lip thick and prominent; the neck is thick and deformed by enlarged thyroid or other enlarged glands; the abdomen is swollen and rather protuberant; the legs small and relatively short. The intellectual powers correspond to the physical—they are slow, inactive, and wanting in strength. Although typical examples of these two forms are met with, many cases consist of a mingling of these types. They present the usual pathological conditions from infancy up. They are subject to attacks of coryza, to scrofulous ophthalmia, to otorrhœa and discharges from behind the ears, to vesicular and pustular eruptions, etc. Slight wounds of the skin are followed by protracted suppuration, by enlargement of the connected chain of lymphatics, and they heal with difficulty. During the first dentition obstinate impetiginous eruptions appear on the face and scalp (milk-crust), and, if the eruptive diseases attack these strumous subjects, severe nasal catarrh, otorrhœa, and unhealthy ulcerations linger long afterward. After the second dentition, the lymphatic glands begin to enlarge, and the *scrofulides*, or scrofulous skin affections, make their appearance—as erythema, eczema, impetigo, ecthyma, and also lupus. Then follow affections of the mucous membranes, which are usually catarrhal, the discharge being yellow, thick, and drying easily, but it is highly irritating, causing about the nose, for example, obstinate eczema. The nose and the ear are special seats of scrofulous suppuration and discharge. The eye is affected by scrofulous ophthalmia, which is remarkable for its persistence and severity, and for the little damage done to the organ, if the affection be appropriately treated. The mucous membrane of the bronchi is a favorite seat of scrofulous inflammation, leading to caseous phthisis and tuberculosis. The lymphatic glands, as has been described, are affected in two modes—by a simple hyperplasia, and by cheesy degeneration and tuberculosis. When the affected glands become very large, forming great bundles, the surrounding connective tissue undergoing inflammation, the change consists in a cheesy degeneration and tuberculosis. Abscesses may form by suppuration of the connective tissue; but these are superficial. When suppuration occurs in the substance of the gland, the skin overlying it is attached, becomes a characteristic bluish-red color, and ultimately breaks, the gland is exposed, and an ulcer is formed, having undermined, irregular, and livid margins. The ulcer thus formed may spread for some distance under the skin, and sinuses extend in various directions, and often burrowing quite widely. Healing of such scrofulous ulcers does not take place until the remains of the cheesy gland are finally extruded, and a large, unsightly, often thick and indurated cicatrix is left. Sometimes the glands enlarge enormously, but do not inflame and suppurate. Such bunches are often seen on both sides of the neck, filling in the whole space from the jaw to the sternum, and extending into the mediastinum. When large numbers of glands enlarge in this way, phthisis is more apt to follow than in the other form characterized by suppuration, according to the author's observation. The most severe of the scrofulous affections are those of the bones and joints, notably fungous arthritis (Billroth). This disease appears most frequently in the knee, but attacks the other joints also, is very chronic in course, and terminates either fatally or in an anchylosed joint. Scrofula also attacks internal parts by affections of the lymphatics, as *tabes mesenterica*, or more frequently as cheesy pneumonia. The nutrition of the body does not necessarily fail. Large ulcers on the surface are not incompatible with very good health and considerable *embonpoint;* but protracted suppuration of bone, disease of the mesentery, etc., make serious inroads on the vital powers, but the mischief induced by the amyloid degeneration, caused by protracted suppuration, is much greater.

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* "General Pathology," Wagner, translated by Drs. Van Duyn and Seguin, New York, 1876, p. 458.

Course, Duration, and Termination.—The course of scrofula is essentially chronic. When one group of troubles disappears, another group comes on the stage. Its course is much influenced by the particular direction taken by the morbid process, whether it attacks the external lymphatics or those of the mesentery, the nasal mucous membrane or the bronchial, etc. In many instances the morbid influence expires about the period of puberty; in others at this period phthisis develops. During the course of scrofula, general miliary tuberculosis may come on, or the protracted suppuration may cause amyloid degeneration of important internal organs, or a tuberculosis of the cerebellum may arise. So many elements enter into the solution of the problem that the duration can not be very definitely expressed, and the termination is affected by so many possible complications that no exact limits can be set for it.

Treatment.—When acquired, the treatment of scrofula is a slow, difficult, and unsatisfactory procedure. Better results are obtained by prevention when the existence of a scrofulous diathesis is suspected. Preventive measures, which must begin at birth, consist in saving the child from all those evil hygienic influences which are the chief exciting causes. A scrofulous mother should not nurse her child, which should be put to the breast of a healthy and vigorous wet-nurse. When feeding begins, the diet should be properly proportioned, and should not be composed of more than the necessary amount of starchy food. Abundance of plain, substantial, and easily digested aliment should be supplied to the growing child ; its clothing should be arranged to protect the body, allow the limbs free motion, and afford the necessary warmth; confinement in-doors, especially to dark and damp habitations, should be prevented, and, if practicable, a healthy country life should be followed up to puberty, and the educational training should be conducted with reference to these essentials of the bodily training. If scrofula has already appeared under any of its modes of manifestation, the hygienic rules just referred to are even more necessary, but unfortunately are attended with less success. As faulty nutrition is an important factor, our remedial measures should be early directed to improve the assimilative functions. The mineral acids and the bitters are very useful here. One of the most serviceable remedies for promoting constructive metamorphosis is the lactophosphate of lime, which is best administered in the form of sirup. For this may be substituted the "phosphates" in the form of the compound sirup ; but the former is more efficient. Cod-liver oil is of great utility in scrofula, but it is better to reënforce the oil with the lactophosphate of lime. If suppuration is going on, the sulphides, according to Ringer, may be depended on to secure the rapid closure and healing of the surface ; but the author regrets to say that he has not succeeded so well with these remedies. If anæmia is a marked feature, the chalybeates are useful. The author finds the sirup of the iodides of iron and manganese a very efficient preparation. Iodine has had, since its first discovery, considerable repute as a remedy for scrofula, but this, originally derived from observation of its effects on simple goitre, has not been confirmed by further experience of its use in the enlarged glands of scrofula. While this is true, it is also a fact that the iodides of iron are more efficient than the other chalybeates. Other remedies advocated for scrofula are the chlorides of calcium and barium, and they deserve a suitable trial in obstinate or protracted cases. A number of topical applications have been proposed. The most efficient in our experience is the ointment of the red iodide of mercury. This can not be used when inflammation has begun in the skin. When scrofulous abscesses form, the pus should be drawn off with an aspirator, and the cavity then injected with tincture of iodine. When there are open ulcers, an excellent application is iodoform mixed with tannin, the powder being blown by the insufflator into all the crevices.

ACUTE MILIARY TUBERCULOSIS.

Definition.—*Acute miliary tuberculosis* is a febrile affection due to the deposit, generally, through the body, of the gray tubercle-granule. It should not be confounded with *phthisis florida*, which is an acute caseous pneumonia.

Causes.—The gray granulation, or miliary tubercle, consists of a fine reticulation of fibers, with a mass of epithelioid cells and granules, and the *bacillus tuberculosis*—the specific element. In acute miliary tuberculosis these minute bodies are widely distributed throughout the system. In the lungs they arise from the irritation of old lesions, from cheesy lymphatics, etc., and they are developed in various organs by the irritation of caseous deposits, of suppuration, of the products of serous and mucous inflammations, etc. Acute miliary tuberculosis is one mode of dying from consumption. That the gray granulation is deposited throughout the body under the influence of certain kinds of irritation, it is necessary that a peculiar vulnerability of the constitution exist—in other words, that it be of the scrofulous type. These deposits of miliary tubercle may occur at any age, but most usually from puberty to middle life.

Pathological Anatomy.—In the brain, miliary granulations develop from the endothelium of the lymph-spaces, and are therefore found chiefly in connection with the pia mater. They occur also in the other membranes, and in the choroid. In the lungs they are contained in greater numbers than elsewhere, and are usually associated with and dependent on other changes in these organs. Nevertheless, both lungs may be infiltrated throughout with the gray granule, when free from any source of irritation. In that case the infection is found to proceed from some other source—from the bronchial glands, genito-urinary tract, or elsewhere. In addition to the tubercular deposition, the mucous membrane of the bronchi is generally hyperæmic, and the congestion increases from the main bronchi downward. There is also increased secretion, the mucus having a somewhat adhesive and viscid character. Miliary granules are quite abundantly distributed in the pleura and peritoneum, as in the pia mater. The liver, spleen, and kidneys, and the mucous membrane of the intestinal canal, are also more or less infiltrated. About the site of each granulation there is a patch of hyperæmia, due to the presence of an irritating material. As so many organs are simultaneously invaded, it follows that their functions must be disordered. As the new formation develops from the vessels, some serious changes might be expected in the composition of the blood. Although not adequately studied, enough is known to show that the blood is much altered. In the lungs, hypostasis takes place, and in various dependent situations the blood transudes. The blood itself is dark, and not readily coagulable. The heart is soft and flabby and its tissue easily torn. The spleen is also enlarged, the pulp much increased, and of a dark-brown color.

Symptoms.—Acute miliary tuberculosis may arise in the course of phthisis, when, therefore, are exhibited the phenomena of a new, sudden, and general infection in addition to the previously existing malady. It may begin in those who have apparently good health, because the source of infection is dormant. It is with the latter class that we have to deal here; the former have been sufficiently considered in the chapters on phthisis. As the symptoms of pulmonary, or cerebral, or of intestinal disturbance may predominate in different cases, divisions may be made accordingly; but, without refining so far, it will suffice to describe the disease as a whole, referring to these peculiarities in passing. The disease sets in, after several days of general *malaise*, with a chill followed by fever, or there is more or less chilliness for the first day. The fever soon rises to a considerable elevation; there are headache, *tinnitus aurium*, wakefulness, or sleep disturbed by dreams, epistaxis sometimes; the countenance is dull, the eyes heavy, and the prostration is great from the beginning. The appetite is gone, the bowels are confined, but are moved copiously by mild laxatives, and the urine is scanty and high-colored. Soon after the onset of the disease, a short, dry cough, which is very harassing, comes on, but the most important symptom connected with the respiratory organs is a greatly increased rapidity of breathing, the respirations numbering forty, fifty, even sixty per minute. The pulse is correspondingly increased, rising during the maximum to 140, 160, or higher, and falling not below 120. The tension of the pulse is low

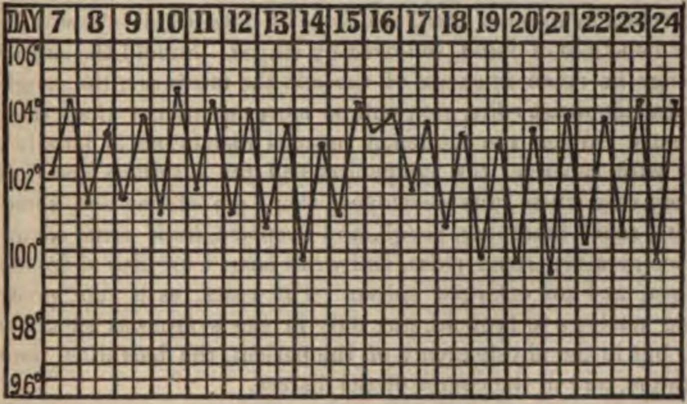

FIG. 56.—Temperature Curves of Acute Miliary Tuberculosis.

(dicrotic) and the action of the heart is feeble. The fever is usually of the remittent type of continued fever, or it has more of the remittent quality of malarial fever, or of hectic. The periods of remissions are charactérized by sweats. The circulation in the extremities is feeble; the finger-nails are blue, the lips and nose have also a cyanotic hue, and the countenance soon becomes dusky. On auscultation, some moist, crackling *râles* are audible over the chest, but there is no special change in the sonority. The difficulty of breathing, noted at the outset, increases and really amounts to dyspnœa. The tongue becomes dry; sordes accumulate about the teeth; food is rejected; the abdomen swells with tympanites; diarrhœa supervenes, the stools being thin and having a light-yellow color; the spleen can be made out considerably enlarged, and occasionally rose-spots, not unlike those of typhoid, appear on the abdominal wall. After the first few days of headache, vertigo, and disturbed sleep, delirium occurs, but at this period the mental disturbance is only at the time of awaking from sleep; by the end of the first week it has become nearly constant. In some cases, so preponderant is the deposit of gray granulations in the meninges of the brain that the symptoms are those of acute meningitis. In a majority of the cases, however, there is delirium of the low-muttering character. As the case progresses, a condition of somnolence comes on; the delirium is less and less active, and the stupor soon passes into coma. When this condition of the cerebral functions is reached, the dyspnœa, before so marked a feature, ceases to affect the respiratory center. When there is little or no deposit of miliary granules in the cerebral meninges, the functions of the brain are disordered because of the high temperature which obtains in this disease. The cerebral symptoms, then, are those of depression—there is a good deal of hebetude of mind, followed by stupor. Should the deposits in the lungs be much in excess of those in the meninges, the cough, the dyspnœa, the moist *râles*, etc., will be more prominent than the head symptoms. When the intestinal mucous membrane is largely infiltrated with tubercle, the tympanites and the diarrhœa are decided. In every case when fully developed, there are stupor and some low delirium, rapid breathing, cough, and dyspnœa, until coma comes on; high temperature, rapid pulse, and weak heart; swollen abdomen and diarrhœa, and an enlarged spleen. The cases, as a rule, present a striking analogy to typhoid, not only in the symptoms as above detailed, but in the physiognomy of the patient, the decubitus, the utter prostration, and in the course of the disease.

Course, Duration, and Termination.—The course of an acute miliary tuberculosis is that of an acute febrile affection. The severity is determined by the extent of the tubercular deposits. The high temperature which prevails at the maxima is a measure of the diffusion of the tubercle-granules, but the fever in turn contributes to the gravity of the case, by inducing the same parenchymatous changes which occur in typhoid. The cases assume somewhat different features, as above pointed out, whether the cerebral, the pulmonary, or the intestinal lesions predominate. The most usual type is that of a severe fever, having bronchial and intestinal complications, and more or less mental disturbance due to high temperature, and hence frequently confounded with typhoid fever. The duration varies somewhat in the different cases, being about four weeks in the largest number, but it may last six weeks or even three months. It is hardly doubtful that death is the invariable termination. The mode of dying is by exhaustion and failure of the heart, by pulmonary obstruction and dyspnœa, and by a gradually deepening coma.

Treatment.—The consideration of the treatment of acute miliary tuberculosis is a rather barren subject, since it does not appear that any remedy has the least influence over the disease. The treatment must hence be symptomatic, and confined to remedies for relieving the abnormal temperature, or for maintaining the power of the heart.

RICKETS.

Definition.—*Rickets* is a constitutional disease of childhood, characterized by a disorder of nutrition in which the growth of the bones is irregular, calcification is imperfect, and deformities ensue. It is also called *osteomalacia, rachitismus, rachitis*, etc.

Causes.—Rickets occurs everywhere, but there are certain parts of the globe where the cases are more numerous than elsewhere, because the conditions are more suitable. Over-populated communities, the people poor, and living in dark and damp habitations, insufficiently fed and clothed, are the social circumstances under which rickets develops. It is common in the great cities of England, and of Europe generally, and rather infrequent in this country. Parry,* it is true, reports that "at least twenty-eight per cent. of all the sick children, between one month and five years old, that have come under his observation during the last three years, have been rachitic." This statement is based on observations in the children's department at the Philadelphia Hospital. Meigs and Pepper, also, of Philadelphia, hold, on the contrary, that rickets is much more common in Europe than in this country. As Gee finds that the proportion of "30·3 per cent. of sick children under two years of age were rickety," and as the proportion for the principal cities of Germany is 25 per cent. for Dresden, 13·4 per cent. for Prague, and 11·1 per cent. for Berlin, this country is rather to be compared with England.† It seems to the author that Dr. Parry's estimate is much too high for this country as a whole, although it may have been correct for the limited area of his observation. The disease, although more prevalent among the children of the squalid poor, also occurs among the well-to-do classes. Certain bodily states of the parents may exert a very baleful influence on the constitutions of their offspring, of which rickets may be regarded as an example. An innate tendency to rickets is a result of marriages of consanguinity, or of those too old, or of the feeble and cachectic. While Sir William Jenner holds that rickets is not inherited, he strongly insists on the influence of the health of the mother on the development of rickets in the child.* All the causes of every kind, which depress the bodily powers of the mother, increase the tendency to the production of rickety children. While the bodily condition of the mother is much more intimately concerned than that of the father, the effect of any given cachexia is much more certain and disastrous when both parents are affected. The rickety constitution may also be inherited. Numerous illustrations of this fact have been collected, and it is generally admitted by authors, but is denied by Jenner. After birth, the hereditary tendency is brought into an active condition by faulty alimentation and unhygienic surroundings. Rickets also occurs in the inferior animals.† The recent observations on "the influence of certain specific irritants upon osteoplastic tissue" have thrown great light on the production of rickets. These specific irritants are phosphorus and lactic acid. If to the action of these, when introduced into the economy, is added a deficiency in the amount of lime-salts contained in the food, or an inability to appropriate that received, there will be produced the state of rickets. Lactic acid is abundantly formed in the intestinal canal of the infant, and acts as an irritant of the osteoplastic tissue, while at the same time it is a solvent of the lime-salts, and thus effects their elimination.‡

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* "The American Journal of the Medical Sciences," January, 1872, "Observations on the Frequency and Symptoms of Rachitis," etc., by John S. Parry, M. D., etc.

† Senator, in Ziemssen's "Cyclopædia," vol. xvi, article "Rickets."

Pathological Anatomy.—The distinctive lesion of rickets is a peculiar alteration of the osseous tissue of the body. The long bones are thickened at their epiphyseal extremity; the bones generally are softened, the flat bones are thickened; various deformities result from the action of mechanical causes, as, for example, deformities of the chest, distorted spine, bent legs, etc.; arrest of growth, not only of the bones themselves, but of all associated parts; related lesions in the pericardium, lungs, and capsule of the spleen; and morbid alterations in the nutrition of the brain, spleen, liver, lymphatic glands, and muscles, etc. (Jenner). Besides these changes, the bones are found in a highly hyperæmic condition, which extends to the periosteum, subperiosteal tissue, and the medulla. The most characteristic changes are those occurring at the junction of the epiphysis with the diaphysis. Calcification of the proliferating cartilage corpuscles goes on irregularly, and the medullary spaces extend beyond the line of calcification. Hence the epiphysis contains cartilage irregularly interspersed in the ossified portions, and the medullary spaces are irregularly bordered by cartilage and by bone. The periosteum is equally changed. Besides an intense hyperæmia, already mentioned, this membrane is much thickened, closely adherent to the bone, and its cellular elements, rapidly proliferating, are being converted into bone-cells. When flat bones are cut across, they are seen to be highly congested, and present a reticulated structure under the periosteum (Senator). The result of these changes is, that the bones are so soft that they can be easily cut, and bent with a slight force. Chemical examination has disclosed important changes.* When the disease is far advanced, the animal matter does not furnish chondrin or gelatin, and gluten has been obtained from it. Jenner finds that while the bones of healthy children yield thirty-seven parts of animal and sixty-three of mineral substances, the bones of rickety children yield about seventy-nine parts of animal and twenty-one parts of mineral matter. Besides the alterations of bone, which are essential, there occur lesions in other organs, some of which are accidental, as the intercurrent diseases ; and others seem to have the relation of effect, as chronic diarrhœa, enlarged mesenteric glands, fatty degeneration of the liver, and enlarged spleen.

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* "Medical Times and Gazette," May 12, 1860, "A Series of Three Lectures on Rickets."

† "Die Rachitis bei Hunden," von Dr. W. Schütz, Virchow's "Archiv," Band xlvi, s 350.

‡ Senator, *op. cit.*

Symptoms.—Rickets begins during the intra-uterine life, and the characteristic changes have been recognized in the fœtus. The usual period of its first symptoms is from the fourth to the seventh month. It is a disease of early life. The cases occurring within the first and second year greatly exceed all of the subsequent life. When the initial symptoms begin, there is a period of several months during which the nature of the case may remain in doubt. The first symptoms are connected with the organs of digestion, and are such as may arise during the course of many chronic diseases. It is observed that the child wastes, but this change is attributed to indigestion, there being more or less diarrhœa and vomiting, the stools and the matters vomited having an acid reaction. The stools are also light in color, because of the absence of bile, and have an odor of decomposition. The appetite is wanting entirely, or is capricious, and vomiting is frequent. Besides wasting, the child grows dull, listless, and peevish ; there is some fever present, and intense thirst is experienced, the child swallowing enormous quantities of water. If the child has begun to walk, it soon becomes too feeble, and prefers to sit or lie quietly, and is equally indisposed to any exertion as to any amusement. Pains in the limbs, especially about the joints, are complained of. The pulse is quick and irritable, and the superficial veins are swollen. The anterior fontanelle remains open and does not diminish in area. These symptoms do not indicate the nature of the disorder which is now developing, but certain signs of high significance make their appearance after a variable period of intestinal troubles and impaired nutrition. To Sir William Jenner we owe the credit of having emphasized the importance of these symptoms. The first is profuse perspirations of the head, neck, and upper part of the chest, appearing chiefly while the child is asleep, but at the same time the abdomen and extremities are dry and hot. The next symptom is a feeling of burning heat, especially in the lower limbs, impelling the child to kick off the covering and keep the legs exposed to the external air in cold weather. The third symptom is tenderness of the whole body. The rickety child does not play and toss its limbs about in all directions, but it keeps as motionless as possible, and cries out when it is taken up, or moved, or pressed on. At this period, also, the urine is abundant, and deposits a copious sediment of the lime salts. The child at this period begins to have a peculiar, a characteristic appearance. It is languid, wasted, its countenance wearied, depressed, and aged, the face has grown broad and square, the hair is thin, dry, and dead, the fontanelle is open widely, the muscles are wasted and flabby, and seem unable to support the body erect, the head sinks between the shoulders, and the abdomen is swollen and protuberant. Now appear the changes in the bones which unmistakably indicate the nature of the case. The extremities of the long bones swell and have a knobby appearance; they yield to the weight of the body or the action of the muscles and bend, those of the lower extremities forward and outward and the femurs forward, and, if the child is walking, outward also. At a more advanced age, the curvature of the lower limbs is different; the knees approximate by bending of the femur and tibia in a curve whose concavity is toward the middle line of the body, and the feet are turned away from each other, so that the child walks on the ankle and inner side of the foot; or the bending is in the opposite direction, both limbs bent like a bow, the child walking on the outer surface of each foot, and the knees widely separated. The spine-curves are determined by the child's walking or not walking. In the former, the natural anterior curvature of the cervical spine is greatly exaggerated; the face is turned upward and the head falls back, and if the muscles are very weak the head is not supported by the neck-muscles, but flops about idly. The other, or posterior curve of the child in arms, commences at the first dorsal and extends to the last dorsal. It may be so great as to be mistaken for angular curvature, and Jenner proposes to differentiate by simply extending the child; but, in old cases, the vertebra and intervertebral disks have undergone permanent changes and can not be moved. Lateral and outward curvature of the spine also takes place ; but these forms are less common, because those that are usual are mere exaggerations of normal curves. Important changes occur in the formation of the thorax. The ribs, being softened, yield to the atmospheric pressure, the sternum is projected forward, thus increasing the antero-posterior diameter of the chest. The ribs are bent posteriorly to an acute angle, and a groove is formed along the junction of the ribs with their cartilages, extending from the first to the ninth or tenth rib, but farther down on the left side. Owing to the position of the heart, the chest-wall of the præcordial space, supported also by the liver, spleen, and stomach, does not recede and hence is apparently more protuberant. Similar curves occur in the upper extremities, but they are determined by the age and the muscular actions imposed on these members. The head of the rickety child appears larger than that of a healthy child of the same age ; but this is only apparent and not real, the difference being due to the wasting of the face and neck in the former. If the rickety child is under two years, the fontanelle, which normally closes by this time, is widely open, and remains open till the third year or longer. The vertex has a flat shape, the forehead is large and square, and the parietal bones are expanded. The bones of the face—the upper jaw and the malar—cease to grow, while the frontal and ethmoidal sinuses expand, and hence the greater prominence of the latter. The process of dentition is either delayed, or it is entirely arrested, or the teeth, if formed, decay and fall out.* The pelvis, as the chest, acted on by the weight of the body and by the muscles attached to it, is deformed in various ways. The sacrum and pubis may be approximated, or the iliac bones may be distorted inwardly, or the outlet may be changed in form and narrowed by the sacrum bending forward. The gastro-intestinal disorders, which precede the osseous changes, continue during the development of the latter. Emaciation goes on at the same rate, the abdomen enlarges still more, the muscles waste and grow weaker, there is less and less disposition to voluntary exertion, the perspirations are more free, the thirst increases, the bowels become more irregular and the evacuations more unhealthy, containing little or no bile, are fetid, the food often passing unchanged. The pains in the bones increase in severity, and their growth ceases entirely. Progressing in this way, after a variable period, the case is terminated by some intercurrent malady, or by the development of some one of its natural sequelæ, or by restoration to health.

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* "Ueber Osteomalachia und Rachitis," von Dr. F. Ruloff in Halle, Virchow's "Archiv," Band xxxvii, s. 433.

Course, Duration, and Termination.—Cases of rickets of so acute a character as to run through their course in a few weeks have been described. In its ordinary form, rickets is an essentially chronic malady, and lasts from months to years, often many years. When the disease begins very early, the changes are more extensive and severe; but those cases are more slow in progress which begin during or subsequent to the second year, and they are hindered in growth by more or less prolonged periods of improvement, during which the bone affection subsides and the intestinal disorders cease for the time, to be resumed when the exacerbations come on. Those cases beginning after the first dentition pursue a milder course, and, if properly managed, end in recovery, but with the deformities and arrested growth of the period of the disease at which arrest occurred. Recovery may take place in those cases occurring the first year of life. When such a favorable course is to be pursued, the teeth, which had been tardy in making their appearance, come through and do not decay, the swelling of the bones subsides, the appetite improves, and the nutrition becomes more active. Various complications arise. Among the most common are catarrh of the bronchial tubes, broncho-pneumonia, capillary bronchitis, congestion of the lungs, and pleural effusion. Jenner strongly insists on the dependence of *laryngismus stridulus* on rickets, or a rickety constitution. The gravity of slight affections of the thoracic organs is much increased because of the diminished capacity of the thorax. Enlarged spleen is present in two thirds of the cases proving fatal. Enlarged lymphatics also may be associated with it, and important changes in the blood take place, a very severe anæmia resulting. Chronic hydrocephalus may also occur as a complication, and death is not unfrequently caused by convulsions. Protracted diarrhœa, ulceration of the intestine, and amyloid degeneration of organs, may also appear during the course of unfavorable cases.

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* Dr. Samuel Gee, "St. Bartholomew's Hospital Reports," vol. iv, 1868, p. 69, "On Rickets." He gives the case of a boy of three years, who had cut only eight teeth.

Diagnosis.—When rickets is fully developed, a question of diagnosis can scarcely arise. The only disease with which it may be confounded is inherited syphilis. Rickets does not appear, as does syphilis, during the first days of life. The "snuffles" and cutaneous lesions do not belong to rickets; enlargement of the epiphyses of the long bones does not belong to syphilis. Local deformities, which may simulate the changes wrought by rickets, are distinguished by the fact that the latter are general and not local.

Treatment.—The most important remedies for rickets are hygienical and dietetic. Good air, warm clothing, daily bathing, and a nutritious diet, are essential. If the child is nursing, the milk of the mother should be carefully examined. If she is the subject of syphilitic infection, or of a cachexia, the child should be removed, although the milk may seem to be entirely healthy. No rickety child should be "raised by hand," if practicable to avoid it. If, however, it can not be nursed, a proper diet becomes then a subject of high importance. Good cow's milk, diluted by one third to one fourth of lime-water, is the most suitable aliment. In the absence of this, condensed milk may be substituted. Should these disagree, as shown by the passage of a great deal of casein in the evacuations, barley-water with one fourth cream added is an excellent substitute. The various substitutes for mother's milk or infant food, offered for sale, are of doubtful propriety, since they usually contain an excess of starchy food, or are prepared on false principles, or based on theory. The points to which medicinal treatment should be directed are the disorders of digestion, the acidity of the evacuations, the absence of bile, and the waste of the lime salts. Lime-water should be given freely with the milk, or carbonate of lime in small quantity may be stirred in the milk. Pepsin in full doses is highly serviceable, and, if there are vomiting and diarrhœa, it may be given with bismuth. Pepsin, with diluted muriatic acid in small quantity, is also useful, the acid acting the part of an anti-ferment, and preventing the formation of lactic acid. Brandy, reënforced as to its astringency by a few drops of tincture of catechu, is a most efficient remedy also, both to counteract the depression and to act as an anti-ferment and an astringent. Cod-liver oil is the most efficient remedy against the constitutional condition. Moreover, cod-liver oil improves the digestion and changes the character of the evacuations. It may be given in an emulsion with lime. The dose should not exceed half a drachm to one drachm, three times a day, but it should be kept up faithfully for a long time. Small doses of iron, the carbonate saccharated, the most easily digested, or the acetated tincture, or the bitter wine of iron, should be persistently administered.

LYMPHADENOMA.

Definition.—By *lymphadenoma* is meant a dyscrasic affection, characterized by enlargement of the lymphatic glands and of the spleen, and by progressive anæmia. It is also called *Hodgkin's disease*, because it was first described by Dr. Hodgkin in 1832,* and is known as "malignant lymphoma," "lympho-sarcoma," the name given it by Virchow, and "pseudo-lukemia," as named by Cohnheim.

Causes.—Little is known as to the influences producing the disease. It is not hereditary ; it may come on without obvious cause in an individual in apparently perfect health ; it is three times as frequent in males as in females, and is more common in youth and old age than in the middle period of manhood, but it may occur at any age.

Pathological Anatomy.—The changes peculiar to this disease are found in the lymphatics and in the spleen. In advanced cases, all the glands of the body, superficial and deep, are diseased, and the adenoid tissue in the course of the lymphatic vessels takes on an overgrowth. The cervical, axillary, inguinal, retro-peritoneal, bronchial, mediastinal, and mesenteric are in turn affected, and in the order named. Usually both sides, but sometimes only one side, is affected. The size of the glands affected ranges from a filbert to a hen's-egg, and when a group of glands is enlarged to the maximum the whole collection forms an immense tumor, which may have the dimensions of a child's head. At first each gland is separate and freely movable; at length the whole group forms a solid mass; but other glands in other situations may still remain mobile. The growth may ultimately penetrate the capsule and extend into surrounding tissues, and may even perforate a vessel. The solidification of a group of glands is also brought about by inflammation of the surrounding connective tissue. The pressure of the enlarging glands may cause atrophy of neighboring structures and interfere with the functions of organs. Two kinds of changes are noted in the glands: some are hard and others soft, but those which have been soft may become hard. Sometimes it is the large, sometimes the small, glands that are hard. On section of an affected gland, the difference between cortical and medullary parts has disappeared; the color is whitish or grayish, with here and there a spot of hyperæmia. The soft glands contain a great quantity of lymph-corpuscles (or cells strongly resembling them), which gradually displace the septa of the gland, and thus give to its cut surface an homogeneous appearance. In the harder glands, the firmness of structure is due to the development of fibroid tissue, which takes place in the septa, in the reticulum, and in the walls of the capillary vessels. Finally, the cells atrophy and disappear before this growth of fibrous tissue. The spleen is enlarged in three fourths of the cases, but slightly enlarged in many of these, the increase in size being due to simple hypertrophy in a few instances, and to disseminated growths in the majority. These growths may be the size of peas, distributed through the organ, or may occur in larger nodules, looking like suet, as Hodgkin was the first to say. These masses are not inclosed in a capsule, but are surrounded by compressed splenic pulp. They do not often pierce the capsule of the spleen, but, if large and numerous, do compress the splenic pulp, which atrophies.* These splenic growths correspond closely with the growths in the lymphatic glands, and consist of the same cells and fibroid tissue; and infarctions are also encountered.† In some cases, the marrow of bones has undergone changes; it becomes converted into a reddish-gray, soft, almost fluid material, due to the predominance of lymphoid cells, and other and larger cells, with compound nuclei. This alteration of the marrow of bones is not unlike that which occurs in leucocythemia. The large follicles at the base of the tongue enlarge to a considerable extent, and the adenoid tissue of the intestinal mucous membrane and of the tonsils takes on the same kind of change as the lymphatic glands. One tonsil may ulcerate, while the other is enormously enlarged. The changes occurring in the adenoid tissue of the solitary glands and of Peyer's patches may result in great thickening of the intestine walls, but do not encroach on the lumen of the bowel.[*] The liver is invaded in a considerable proportion of the cases by minute lymphoid growths, varying in size from a pin-head to a pea, and having the same composition as those of the spleen. In other cases the adenoid tissue is not disseminated in isolated masses, but accompanies the portal vessels occupying the interlobular spaces, and sending processes into the acini. One third of the liver may be thus occupied.[†] Fatty degeneration may coincide with the lymphoid disease in the liver, adenoid growths occur in the kidneys also, and chiefly in the cortex. The growths are of small size—from a pin's-head to a pea—and are disseminated in the inter-tubular spaces. They cause atrophy by pressure, and initiate parenchymatous degeneration with the usual consequences. The same growths are rarely found in the ovaries and testes, and often in the thymus. The lungs may be attacked by contiguity of tissue from the diseased bronchial glands, or by the vessels. The growths found in the lungs are small, grayish, and firm, and are often mistaken for tubercles (Gowers[‡]). More or less effusion occurs in the thorax, and sometimes, but rarely, lymphoid growths are found in the sub-pleural tissue, and in the substance of the diaphragm. Sometimes the heart is small; again it is far advanced in fatty degeneration; only rarely have the characteristic adenoid growths been detected in the substance of the organ. Murchison[§] records an adenoid growth of the dura mater above the foramen magnum, and Mosler one above the foramen opticum.

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* "Medico-Chirurgical Transactions," vol. xvii, 1832, p. 68, "On some Morbid Appearances of the Absorbent Glands and Spleen," by Dr. Hodgkin, presented by Dr. R. Lee, read January 10 and 24, 1832.

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* Virchow, "Die Krankhaften Geschwülste," zweiter Band, s. 735, Fig. 203.

† Langhaus, Virchow's "Archiv," Band liv, s. 512.

Symptoms.—There are two groups of symptoms: those due to the disease, *per se;* those due to the interference by the growths in the functions of various organs. As regards the first group there are two distinctive symptoms—the enlarged glands, and the anæmia. The cervical lymphatic glands are, in a majority of cases, the first to enlarge, and the others, as a rule, follow in the order which has been already given. In a few instances a febrile attack accompanied the initial trouble in the glands; in other cases the irritation of some glands, temporarily and from trivial causes enlarged, has led to the development of the general disease, but some kind of predisposition must have existed. The enlarged glands are firm or soft, and are painless unless nerves are pressed on. Anæmia may begin and be considerably advanced before the glandular enlargements, but it usually succeeds to them. The anæmia of lymphadenoma is like the anæmia of any cachexia. The functions generally are depressed, and we have, in addition, the weak heart, the breathlessness on exertion, and the pallor and feebleness belonging to this state. The number of white corpuscles in the blood is not in excess of the normal in the majority of cases, and is never considerably above normal in any case. The white-blood corpuscles are small, as a rule, and vary in size. The red corpuscles are reduced in number, and in some cases the number of small red corpuscles is large. According to Gowers, the red corpuscles, as counted by means of the hæmacytometer, may descend to sixty per cent. of the normal in a subject having still some color. Fever occurs in about two thirds (Gowers) of the whole number of cases. Fever may be present, also, as a symptom of some intercurrent febrile affection; but it is a part of the morbid process in young subjects. Although the course of the fever is irregular, three types are known: a continuous type with slight diurnal variations; a remittent fever, hectic in character; and a paroxysmal fever, with intermissions of entire cessation of fever for several days. The symptoms due to pressure are as various as the organs pressed on. The enlarging cervical glands and thyroid press on the carotids and jugulars, interfere with the intra-cranial circulation, producing at one time cerebral anæmia, at another time passive cerebral congestion. Deglutition may be interfered with by pressure on the pharynx and œsophagus, voice and breathing by pressure on the larynx and trachea. The glandular swellings in the chest produce all the symptoms of intra-thoracic tumors, by pressure on the cardiac branches of the sympathetic, on the recurrent laryngeal, on the pneumogastric and phrenic, on the great venous trunks, on the arteries, and on the trachea, bronchi, and œsophagus. Within the abdomen these tumors may compress the aorta and give rise to the symptoms of aneurism, the stomach, and cause nausea and vomiting, the portal vein and hepatic duct, and induce ascites and jaundice, the principal nerves, and arouse pain, and the great veins, producing œdema of the lower extremities. To enumerate all the symptoms which may be excited by the pressure of these enlarged glands would be to summarize the symptoms which may be expressed by any disordered organ.

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[*] Moxon, "Transactions of the Pathological Society," 1873, p. 101. Murchison had made the same observation in a case of the same kind, "Pathological Transactions," 1870.

[†] Wilks, "Guy's Hospital Reports," 1865, "Cases of Lardaceous Disease and Allied Affections," p. 128, "Peculiar Enlargement of the Lymphatic Glands."

[‡] Dr. W. R Gowers, Reynolds's "System," vol. iii, American edition, article "Hodgkin's Disease." The author has to express his indebtedness to this elaborate and exhaustive memoir for valuable information.

[§] "Transactions of the Pathological Society," 1870, p. 372. A full history of the disease follows.

Course, Duration, and Termination.—The course of lymphadenoma is chronic. The average duration of fifty cases collected by Gowers was nineteen months; of eighteen cases, the duration was less than one year; of fifteen cases, between one and two years. In the only case which the author has had in his own charge, the duration was two years. In all cases the initial glandular enlargement—cervical usually—is followed after a certain interval by the general affection of all the glands. There may be quite an interval, sometimes years, however, between the local and systemic affection. The course of the disease may be influenced by complications. The anæmia may induce various acute inflammations—erysipelas, superficial abscesses, etc. Phthisis may occur, as in the author's case. Death is usually due to exhaustion, but it may be caused by pressure on the trachea and asphyxia, on the œsophagus and starvation, on the jugular veins, carotids, and convulsions and coma. Certain intercurrent affections may cause death, as pneumonia, œdema of the lungs, pleuritic effusions, etc.

Diagnosis.—The maladies with which Hodgkin's disease may be confounded are leucocythemia, with splenic and glandular changes, and scrofula. In splenic, glandular leucocythemia the changes in the glands succeed to those in the blood, whereas the glandular enlargement is the initial fact in lymphadenoma; and, further, in the latter, the relative proportion of white corpuscles is not increased in the majority of cases. From scrofula the distinction is made by the number, extent, and volume of the glands in lymphadenoma, by the extension of the enlarged glands over the body, by their permanence, by the anæmia, and by the pressure symptoms which affect so many organs. In scrofula the enlarged glands are found in one situation, and usually about the neck they suppurate ; the symptoms are limited to the affected part, and there is neither anæmia nor pressure symptoms.

Treatment.—Recent experiences by Billroth and Czerny * have demonstrated the curability of lymphadenoma by the internal and parenchymatous use of arsenic. Fowler's solution is usually employed, the dose by the stomach being increased to ten, fifteen, or twenty minims, thrice daily, according to the forbearance of this organ, and from one to five minims in distilled water injected into the enlarged glands. Wunderlich has reported a case improved under the use of iodide of potassium. The sirup of the iodides of iron and manganese has seemed to do good by improving the cachexia. Cod-liver oil is certainly useful as a nutrient and tonic, but it can not be regarded as curative. Electrolysis has been much commended, but thus far no successful cases have been reported. It is obvious that this plan of treatment could only be used in the case of the first enlarged glands. Extirpation of the diseased glands, when but few are affected, has been done, but, as great uncertainty must exist in regard to the nature of the malady and the relation of those glands first attacked to the subsequent development, the surgical operation can not be considered a desirable expedient.

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* "Wien med. Wochenschr.," No. 2, 1881.

ACUTE RHEUMATISM.

Definition.—*Acute rheumatism* is a constitutional disease characterized by fever, inflammation of the joints occurring in succession, and by a tendency to attack the peri- and endocardium. It is frequently called *articular rheumatism, rheumatic fever, polyarthritis rheumatica,* etc.

Causes.—The vice of constitution belonging to rheumatism may be inherited, but it is not possible to indicate its character.* There are three types of bodily conformation in which rheumatism occurs: the pale, thin, and anæmic subject; the robust and vigorous individual with an inherited tendency; and the obese, often given to the consumption of malt-liquors and having a form of acid indigestion (lactic?). Acute rheumatism is most frequent in youth and early manhood, rarely occurring before seven and after fifty. It is more frequent in men than in women, not because of a greater susceptibility to the disease, but because men are more exposed to the influences producing it. The liability to the disease is increased by having attacks, and a longer interval usually separates the first and second seizures than the second and third. On the other hand, the susceptibility to rheumatism lessens with increase of years. Certain diseases dispose to attacks of acute rheumatism: thus during the stage of desquamation of scarlet fever, and in the puerperal state, attacks in all respects the same as ordinary rheumatic fever may occur. The seasons of greatest prevalence are winter and spring, and the occupations most favorable are those in which there is the most frequent exposure to inclement weather. Protracted stay in damp apartments, lying between damp sheets all night, exposure of the body to cold and wet when in a heated and perspiring state, are fruitful causes of attacks, the predisposition already existing. The frequency with which rheumatic attacks follow exposure to cold, to chilling the superficies of the body, is a very striking fact. Senator † ingeniously supposes that the irritation of the peripheral fibers of the centripetal nerves excites the vaso-motor and trophic centers into abnormal activity. Various facts go to prove that a condition of the joints not unlike rheumatism is brought about by certain diseases of the spinal cord and injuries of nerves.‡ As, during muscular exercise, lactic acid and the acid potassium phosphate are produced, and as an excess of acid is a fact in rheumatism, and, further, as sudden chilling of the body stops the elimination of those acid products, which therefore accumulate, there would seem to be a necessary connection between these states. The agency of lactic acid in producing rheumatism seems further strengthened by the fact, first observed by Richardson, that the injection of lactic acid is followed by endocarditis, and its medicinal administration in diabetes has in various instances apparently caused a rheumatic inflammation of the joints. This chemical theory, originally proposed by Prout and supported by Richardson's experiments, has received a severe blow in the denial by Reyher * that the injection of lactic acid is followed by endocarditis, as affirmed by Richardson, or that an accumulation of the acid in the blood is a cause of rheumatism, as suggested by Prout.

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* Notwithstanding the agency of a damp climate in causing acute rheumatism, in New Mexico, a remarkably dry climate, this disease prevails largely. Indeed, the author saw, in 1860, what might be regarded as an epidemic.

† Ziemssen's "Cyclopædia," vol. xvi.

‡ "Injuries of Nerves and their Consequences," S. Weir Mitchell, *op. cit.*

Pathological Anatomy.—The changes in the joints are slight as compared with the apparent extent of the mischief. The synovial membrane is injected more or less deeply, and the fringes are highly vascular. The membrane has lost its pearly transparency and its smoothness, and is cloudy and granular. The synovial fluid is increased in amount and is changed in character. Instead of being a transparent, homogeneous, viscid fluid, it is thin, watery, reddish from extravasated blood, turbid from the presence of fibrin, and some pus-corpuscles. There is never any considerable amount of blood present in the fluid, except in the case of the hæmorrhagic diathesis, and the quantity of pus is slight unless the rheumatic inflammation is complicated by some other malady. A half-century ago much importance was ascribed to the excess of fibrin in the blood, to the buffy-coat and to the cupped appearance of the clot; but these features of the blood composition are not now considered to have any special significance, besides the excess in fibrin. Garrod states that the quantity of fibrin reaches from four to six parts per thousand. The serum is alkaline, and is free from uric acid and lactic acid. The usual complication of acute rheumatism is inflammation of the peri- and endocardium. The nature of the pathological changes in these cardiac affections is set forth in the articles on these topics.

Symptoms.—For several days previous to the attack of acute rheumatism, the patient complains of muscular soreness, often of neuralgic pains localized in some important nerve; in other cases the patient experiences a good deal of pain, stiffness and soreness of certain joints, and with these joint- and muscle-pains and soreness are associated an impaired appetite, coated, pasty tongue, constipation, etc. The disease may begin abruptly without the prodromic symptoms just described, by a chill, followed by fever, or by a succession of slight chills with fever, the temperature rising to 102,° 103,° or 104° Fahr. There occur also, thirst, a coated tongue, anorexia, and constipation; headache and wakefulness are experienced; and the ankles become painful and can not support the body. Examination of the painful joints discloses the fact that they are tender, hot, swollen, and red, and every attempted movement produces exquisite suffering. On the same day, or certainly the next day, other joints are affected, and those first attacked get a little easier and the swelling slowly subsides. In the first attack the larger joints are affected almost entirely, but in succeeding attacks the smaller joints, especially of the hands, suffer severely. The joints first attacked and getting well may be seized upon again, and in turn most of the joints of the body are affected. By the end of the first week, a number of joints, six, ten, even twelve, may be inflamed. The joints most frequently visited are the ankle and knee; next, the shoulder, elbow, and wrist; then the hip and fingers, and finally the spine, the toes, and the lower jaw. Even the crico-arytenoid articulation may be attacked (Senator). The disease seems to pursue a certain order in its visits to the articulations—first touching at the right ankle-joint, then flying over to the left, then the right knee is reached, afterward the left (Garrod). The suffering imposed by a rheumatic seizure is very great in any case, but is the greater the larger and the more numerous the joints inflamed. When the spine is attacked the pain and inconvenience are at the maximum, for no movement of the body can be attempted, and even breathing is painful. The position assumed by the patient is the easiest which his disability will permit; the limbs are half flexed, the foot turned in a little, and the hand extended, the fingers separated more or less widely. So exquisitely tender are the joints, in many cases, that the patients manifest uneasiness when any one approaches the bed; the weight of the bedclothes becomes intolerable; and even the jar of one walking heavily over the floor awakens pain. The joints are red and swollen, and sometimes the tendons and connective tissue about the joints are infiltrated and œdematous. On the other hand, the joint may have a natural appearance and yet be very painful. Even when quite a good deal swollen, the inflammation may subside in a few hours, and attack other joints in a corresponding way.

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* "Zur Frage von der Erzengung von Endocarditis durch Milchsäure-injection," etc., by Dr. Gustav Reyher, Virchow's "Archiv," vol. xxi, p. 85.

This tendency to migrate from one joint to another is the most characteristic feature of acute rheumatism. As the effusion into and about the joint is serous, and as, besides this, only a condition of hyperæmia is present, it is not surprising that such sudden transitions take place. In the mildest cases, with few joints affected, and without complications, the fever is slight, consisting of an exacerbation developing toward evening, and entire freedom from any increased body-heat the rest of the time. In the decided cases, however, there is fever of a somewhat remittent type, the exacerbation coming on in the afternoon. The maximum rarely exceeds 104° Fahr., and the usual temperature is 100° to 101° in the morning and from a half to one degree higher in the afternoon. The range of febrile heat is not uniform; besides the daily variations, remissions and even intermissions take place during the course of the disease. If there should occur a complete intermission, usually there is an exacerbation of all the symptoms with the rise of fever. The termination of the febrile movement is gradual and not by crisis. Now and then a case of remarkable severity is encountered. Violent delirium occurs and a state of hyperpyrexia comes on, the temperature rising to 108°, 109°, and even 111° Fahr., has been noted,* and the rise continues subsequent to death, for a short time. Dr. Ringer observed that this condition came on suddenly in three cases who were doing well. Either delirium followed by stupor or stupor without delirium appeared without any warning, the temperature rose to 111° in one case, and to 109° and 110° in the others, and death ensued in all in a few hours. Quincke, Wilson Fox, and others have reported similar cases, but they are fortunately rare. Delirium, coma vigil, excitement with very high temperature, phenomena not unlike one variety of heat-stroke, occur in the case of spirit-drinkers or the cachectic attacked by acute rheumatism. The rate of pulse is not usually conformable to the temperature curve, because it is accelerated by other causes—chiefly by the pain. There is in acute rheumatism not a hot skin, because of the sweating. This free action of the skin is a part of the morbid process ; it occurs with the joint affection, and subsides somewhat before the latter, and returns with a relapse. The sweat is acid in reaction, and the linen and person of the patient have a strong acid odor. The sweat also contains urea, and formerly was supposed to owe its acidity to lactic acid—a statement which has not been confirmed. As in other diseases characterized by profuse sweating, sudamina appear on the skin. Other eruptions are also sometimes present—urticaria, purpura, herpes, etc. The severe loss by the skin necessarily lessens the quantity of urinary water. The urine is concentrated, strongly acid in reaction, of a deep-red color, and deposits a great quantity of urates and uric acid. The chlorides of the urine are diminished, the sulphates are increased (Parkes), and the urea is also greater than normal in its relative proportion. Albumen is present in the urine in small amount.

Course, Duration, and Termination.—The course of acute rheumatism is much influenced by complications. The most important complication is the rheumatic inflammation of the peri- and endocardium, and of the cardiac muscle. This sometimes is the first symptom, the joint affection appearing subsequently. The author saw in New Mexico cases of rheumatism pursuing this course. The relative proportion of heart cases to those having joint lesions only is stated differently by different authorities, Bouillaud standing at one extreme with fifty per cent., and Chambers at the other with five to seven per cent.* There can be no doubt that great differences exist, and hence no numbers can state the true proportion. The inmates of hospitals have a greater tendency to heart complication than those sick under favorable conditions at home. The existence, then, of the various diatheses and cachexiæ must exert an unfavorable influence over the course of acute rheumatism. Again, youth is a predisposing cause of cardiac complication, a fact which Senator formularizes as follows: "The younger the patient, the greater the risk of his heart becoming affected." Treatment, according to the exhibit of Dr. Dickinson, exercises no little influence over the tendency to cardiac complications, if rightly directed. Comparatively rare complications are bronchitis and pneumonia—the former occurring the more frequently. Pleurisy is still more common because induced by contiguity of tissue, and hence of the left side chiefly, although it may be double. These complicating diseases differ in no material way from the same idiopathic affections. Meningitis has rarely occurred, and doubtless, of the cases reported, most of them were examples of cerebro-spinal meningitis. The natural history of acute rheumatism has been determined thoroughly. The mint-water treatment of Sir William Gull and Dr. Sutton, and the expectant methods of Garrod and of Flint, have demonstrated the course pursued by rheumatism when not interfered with by remedies. The disease manifests a tendency to spontaneous cure about the thirteenth to the fifteenth day, and still more decidedly from the fifteenth to the twenty-first day. The average stay of rheumatic patients in Guy's Hospital, when subjected to the "mint-water treatment," was for males 27·6 days, and for females 26·8 days.† The conclusions arrived at by the advocates of non-intervention have been severely contested by Dr. Fuller and others. In almost the last paper written by the late Dr. Fuller,‡ he has demonstrated the fallacy underlying the observations of the Guy's Hospital clinicians, and has proved the immense superiority of the so-called alkaline treatment. Notwithstanding the disease may be classed with the self-limited, its course is materially abbreviated not only by the alkaline, but by other methods of treatment. The acute stage of a rheumatic seizure, if the first one, is not often terminated in an earlier period than two weeks, and is more frequently prolonged to three or even four weeks. After the first, the subsequent attacks are usually less severe, and the acute symptoms terminate in one to two weeks, and may be prolonged to three. The duration is, however, materially affected, not only by the complications mentioned above and by the treatment, but by the number of joints visited. If more than six joints are visited, the duration of the acute symptoms will not be less than two to three weeks; and, if a dozen joints are one after another brought within the diseased circuit, the duration will be scarcely less than the traditional six weeks. So many factors, therefore, are concerned, that results must be very uncertain which are arrived at without estimating the value of all. Rheumatism is by no means a serious disease if judged from the standpoint of its immediate effects, but it becomes more formidable when the cardiac and other complications arise. The mortality from rheumatism alone does not exceed three per cent.; but the after-consequences of the cardiac lesions are responsible for a great many more deaths. When death occurs during the seizure, it is determined by the condition of hyperpyrexia with delirium most frequently, and alcoholic excess is probably the real cause of this accident in most cases. Now and then a fatal result may be due to meningitis, but more frequently to peri- and endocarditis, with myocarditis. In a very small proportion of cases joints may be permanently damaged by thickenings and deposits, and slow chronic synovitis.

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* Ringer, Dr. Sydney, "On some Fatal Cases of Rheumatic Fever, accompanied by a Very High Temperature of the Body," "Medical Times and Gazette," October 5, 1867.

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* But Dr. Fuller, in his "Treatise on Rheumatism," puts the proportion of heart complications at one third, after examination of many statistics (third edition, pp. 258–284).

† "Guy's Hospital Reports" for 1865, "Cases of Rheumatic Fever, treated for the most part with Mint-Water," collected from Dr. Gull's case-books by Dr. Sutton.

‡ "The Practitioner," vol. ii, p. 129.

Diagnosis.—A well-developed acute rheumatism can hardly be mistaken for any other disease, but there may be difficulty in differentiating it from pyæmia, rheumatoid arthritis, acute general gout, urethral rheumatism, and hysterical joint. Pyæmia differs from acute rheumatism in the type of fever, the periodical sweats, the jaundice, the prostration, and the suppuration and disorganization of joints. Acute rheumatoid arthritis is stationary, and is free from constitutional disturbance, from sweats, and from cardiac lesions. From acute general gout it is distinguished by the fever, the sweats, and the cardiac mischief. Urethral rheumatism attacks one joint, the ankle or wrist, most usually, does not migrate, is slower to recover, is unaccompanied by fever, and is coincident with a urethral discharge. Hysterical joint is without swelling or change of temperature, is exquisitely sensitive when the attention is fixed on it, and can be handled even roughly when the attention is directed to other objects, and is accompanied with other hysterical manifestations.

Treatment.—Opinions are still greatly divided as to the best treatment of acute rheumatism. As controversial discussions do not enter into the scope of this work, the author confines himself to the expression of his convictions. The alkaline treatment has been a real and important advance, but the general conception of what is meant by it is singularly cloudy. Senator gravely proposes the use of the soda-salts because of the supposed toxic effects of the potash-salts on the heart. "By the alkaline treatment," says Dr. Fuller, "I mean a plan of treatment in which alkalies play an important part, but which consists not only in the administration of alkalies, but in the careful regulation of the secretions, the strictest attention to diet, and the administration of tonics, such as quinine and bark, as soon as the patient can bear them." In the treatment by alkalies, the object to be accomplished is, to effect the alkalinization of the secretions, and any result less than this will prove a failure. Fuller gives not less than an ounce and a half of the alkaline carbonates, either alone or in combination with a vegetable acid, during the first twenty-four hours of the treatment. Two drachms of bicarbonate of potassium are given in a state of effervescence by means of an ounce of lemon-juice, or a half-drachm of citric acid, in four ounces of water, every three or four hours. If the bowels are torpid, as is usual, two compound cathartic pills are administered. If the urine no longer exhibits an acid reaction after twenty-four to thirty-six hours, the quantity of alkali is diminished one half. If the urine continues alkaline at the end of another twenty-four hours, three drachms of alkali only are given for the next twenty-four hours; and on the fourth day, if the alkalinity of the urine persists, the form of the medicine is changed, and a tonic is added to the alkali, giving three grains of quinine with a half-drachm of potassium bicarbonate three times a day. Aperients are given as required, and opium as little as possible, and only when there is excessive irritability. The diet is restricted to milk, beef-tea, or broths, barley-water, etc., and under no circumstances solid food until the tongue is clean and convalescence established. The patient is kept between sheets rather cool, and the heaping up of extra blankets on the bed is not permitted. We have been thus full and minute in describing Dr. Fuller's method, from a conviction of its great value in appropriate cases. It relieves the pain quite speedily, shortens the duration and lessens the violence of the disease and prevents heart complications. The average duration of the cases thus treated is put by Dr. Fuller at *eleven days*. Of 439 cases subjected to this plan there was not a fatal case; only a little over two per cent. suffered with a cardiac complication. Dr. Dickinson's statistics are not less striking. Of 161 cases, 113 were subjected to some other than alkaline treatment, and in thirty-five, or 30·8 per cent., the heart became involved; while only one of forty-eight cases treated with alkalies so suffered.* In the pale, feeble, and anæmic young subjects attacked with acute rheumatism, alkalies are as a rule too depressing, and are followed by a tedious and protracted convalescence. In this class of cases we possess a valuable resource in the tinctura ferri chloridi, first proposed by Dr. Russell Reynolds. This remedy must be given in full doses well diluted with water (℥ ss. of the tincture to six ounces of water taken through a glass tube every four hours). It has a most favorable influence over the progress of these cases, and, as Dr. Anstie pointed out, is very effective as a prophylactic against the disease when an attack is impending. For the acute rheumatism succeeding to scarlet fever, to puerperal fever, etc., it is especially desirable and successful. At the present time no remedy is so universally employed in the treatment of rheumatism as salicylic acid in various forms. The success which attends its use is on the whole remarkable. Now that the enthusiasm which first followed its use in rheumatism has subsided somewhat, a fair estimate of its powers can be made. As it causes very great depression of the heart, and excites irritation of the stomach, its utility is much more limited than was at first supposed. Furthermore, although its action is very prompt, relieving the principal symptoms of the disease in two or three days, the tendency to relapses is very great. In a recent paper by Dr. Greenhow,* we find a most able exposition of the effects and real utility of the salicylates. He finds with others that great immediate relief follows the administration of these remedies, that the temperature declines and with it the pain, but serious toxic phenomena often ensue, and relapses occur. Moreover, the drug in considerable doses depresses the heart, obliterates the first sound, and causes vomiting, tinnitus, hallucinations, etc. Salicin, salicylate of soda, and salicylic acid, to be effective, must be given in sufficient quantity to lower the temperature—a half-drachm of salicylate of sodium every four hours, until the pulse and temperature decline, may be taken as the standard. When the pain and fever subside, the dose may be reduced to a scruple. In the discussion which followed the reading of Dr. Greenhow's paper, the speakers insisted on the persistent use of the remedy to prevent relapses. As the effects of salicylic acid and its congeners are decidedly spoliative, the patient is left in a weak and anæmic state. It is good practice, according to the author's experience, to give the muriated tincture of iron as soon as the reduction of heat and pain is effected, while smaller doses of the salicylates are continued. Dr. Greenhow finds that the blister-treatment is quite as successful as the treatment by salicylates, and open to less objection. The blister-treatment as revived by Dr. Davies, of the London Hospital, consists in the application of armlets, wristlets, and fingerlets of blistering-plaster about the inflamed joint, but not on it, as carried out by Dr. Dechilly. The author has ascertained that an investment of the joint by small blisters, leaving space between them all around the joint for succeeding applications, is a good method. Blisters relieve the pain remarkably, change the reaction of the urine from acid to neutral or alkaline, and prevent complications. With blisters may be combined the excellences of the other plans of treatment. The alkaline treatment is particularly applicable to "the obese, florid, but flabby drinkers of malt-liquors"; the iron-treatment to the pale, delicate, and anæmic young subject; and the salicylic treatment to the vigorous, able-bodied subjects of the inherited tendency or rheumatic diathesis, while blisters may be, with proper precautions, utilized in all forms of the disease and combined with any plan. The complications of acute rheumatism are to be treated according to their character. The most important, because so rapidly fatal, is the condition of hyperpyrexia with coma. Since the remarkable efficiency of the cold bath has been ascertained, better results are had from the treatment of this condition than ever before. Quiet and rest are of great importance. Solid food must not be given the patient until the tongue is clean and the digestion active. Milk, above all things, is the most suitable article of diet.

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* London "Lancet," January 23 and 30, and February 6, 1879.

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* The London "Lancet," May 29, 1880, "Cases of Rheumatic Fever treated with Salicylate of Soda," "Transactions of the Clinical Society."

CHRONIC RHEUMATISM.

Definition.—By *chronic rheumatism* is meant an affection of the articulations, characterized by pain and stiffness, with some swelling, occurring chiefly after middle life, and influenced by atmospheric changes.

Causes and Pathogeny.—The chronic may succeed to the acute form of rheumatism. In all cases of the acute disease the joints remain sore and stiff for a short period after the acute symptoms have ceased; but in a few, owing to the constitutional state, to improper management, too early use of the joints, etc., the articulations remain swollen, more or less tender, and disabled. The case may be chronic from the first. If the predisposition exist, exposure to cold and dampness, working in the water, etc., will develop the disease slowly, and those joints undergo alterations first which are most exposed to injury, and to cold and dampness in the performance of their functions. The changes of structure are not well defined in many instances, because of the fugitive attacks; in others, however, there are plain evidences of mischief done. The synovial membrane becomes cloudy, thickened, and rough, and the cartilages also undergo proliferation of their corpuscles and subsequent thickening. Very little effusion of fluid occurs into the synovial sac. Fatty degeneration of the articular cartilages, erosions of the same, slow changes in the bone, leading to induration and thickening, resulting in a limited extent of motion of the articulation, are also results of the morbid process.

Symptoms.—The trouble is limited to the articulations affected and to the neighborhood. The joint is swollen more or less, and its movements are constrained; it is not red and hot unless some recent inflammatory mischief has been lighted up; pain is felt in the joint spontaneously, and soreness whenever the joint is moved, and acute pain is experienced when there occur changes of temperature and the barometer is falling. Patients soon learn the indications, afforded by their pains, when storms are imminent, or other atmospheric perturbations

The joints are stiff, their movements slow and jerking. As the sheaths of the tendons are thickened by deposits, movements cause more or less creaking, like rusty machinery, which may be audible. In the morning, on rising, movements are particularly slow, rigid, jerking, so that dressing is accomplished with difficulty; use renders them limber and supple. Various joints are affected, as a rule, but the disease does not migrate from one joint to another; they may be affected simultaneously or in turn. The muscular pains, which usually accompany the joint affection, are due to the extension of the disease to the sheaths of the tendons in the neighborhood of the articulations. Myalgia is a frequent coincident affection, and hence it is confounded with the rheumatism.

Course, Duration, and Termination.—Chronic rheumatism is a very chronic disease. There occur but few changes from month to month. Exposure to cold, and especially to cold and dampness combined, increases the pains and the joint changes; and warmth—especially removing to a warm climate—lessens them. Fatigue, manual labor, especially in cold and damp situations, and clothing insufficiently warm, promote the disease. In forming conclusions as to the future course of the malady, these elements must be taken into consideration. A perfect recovery must be regarded as possible only in those cases treated at the outset under favorable hygienic and personal conditions. When deposits have taken place, and the cartilages and synovial membrane are changed in structure, a cure can not be effected. In old cases tendinous anchylosis may result, and, the muscles wasting, the limb will appear much deformed. Chronic rheumatism never causes death, nor does it indirectly abridge life except by depriving the patient of rest and sleep.

Treatment.—The remedies intended to assail chronic rheumatism, from the constitutional side, are numerous, but they accomplish little. Colchicum, guaiacum, conium, etc., formerly so much employed, have no longer any repute as remedies in this disorder. There are, however, a few remedies of real value—cod-liver oil, iodide of potassium, muriate of ammonia, and the lithium salts, notably the bromide. Cod-liver oil should be given with a little ether to assist its digestion, and in the dose of a teaspoonful three times a day after meals. To be of real service, the administration of the oil should continue for many months. If there is anæmia, chalybeates should be given. A course of iodide of potassium, if the general health of the patient is fairly good, often renders important service. It is necessary to give it many months, however. Deposits about joints may sometimes be absorbed during the administration of muriate of ammonia, but, to accomplish anything, prolonged use is necessary. In several cases the author has had excellent results from the bromide of lithium. Under its use the pains ceased, the swelling subsided, and the suppleness of the joints was restored. Local applications are highly important. Frictions of the affected parts with cod-liver oil, after a general warm bath, are an excellent expedient. Warm baths, the Turkish or Russian baths, with local douches, are often, but not invariably, highly useful. The method of friction and movements, known as *massage*, is probably the best of the local means of treatment. Good results are obtained from the baths of the Hot Springs of Arkansas, the warm and hot springs of Virginia, the sulphurous waters of the Licks of Kentucky and of Saratoga, the Michigan springs, St. Catherine's of Canada, and numerous other "resorts" in this country. Mud-baths are also employed on a large scale, for the relief of rheumatism and affections of the skin, in certain parts of Germany. In chronic rheumatism excellent results are obtained from the use of galvanism. A current of large volume and low intensity should be applied to the affected joints to procure absorption of effusions, and the sympathetic ganglia should also be brought within the circuit. When galvanism is to be applied, the positive pole should be placed over the principal nerve-bundles above, and the negative pole brushed over the joint-region. Each joint should be taken up in turn, and the applications be faithfully made, and the electrical treatment pursued for a long time.

GOUT—PODAGRA.

Definition.—By the term *gout* is meant a constitutional malady, inherited, and characterized by the occurrence of paroxysms of severe pain in a small joint—the great-toe usually—due to the presence of uric acid in the blood, and the deposit of the urates in the structures of the articulation. *Podagra* is the Latin name for gout in the foot; *chiragra*, for gout in the hand; and *gonagra*, for gout in the knee.

Causes.—Unquestionably, heredity is the chief etiological factor. The causes which rendered the disease hereditary will, of course, produce the disease anew in those subjected to their operation. As a disorder of the upper classes—of those having wealth, leisure, and the opportunity for indulgence in the pleasures of the table—gout has had a position of distinction. Sydenham consoled himself for his sufferings from gout by the reflection that it is an eminently respectable disease, by which more rich men than paupers, more wise men than fools, are afflicted. But this satisfaction is no longer afforded the victims of this malady. Gout is a result of lead-poisoning, and indulgence in the drinking of beer and other malt-liquors, and it therefore occupies a more humble position than formerly. Men suffer from attacks of gout much more frequently than women, and this fact is as true of inherited as of acquired gout. It is suggested by Garrod (originally by Hippocrates) that the catamenial function acts as a "safeguard," because, when the inherited tendency exists, the outbreaks rarely occur until after the menopause. The chief reason of the comparative exemption enjoyed by women is the difference in habits; when women adopt the meat-eating, and beer- and wine-drinking habits of men, they suffer the same consequences. Gout begins at a comparatively early age, when the bodily predisposition and the habits of life favor its appearance. Paroxysms may begin at fifteen, but when the disease is acquired they are postponed to thirty-five or later. The period of greatest predominance of the affection is from thirty-five to sixty-five, and after the latter age it is less and less common. The habits of the individual are largely concerned with the early production of gout. The drinkers of malt-liquors and wines, especially the sweet wines, suffer early. It is the large consumption of beer which develops the gouty condition in the laboring classes. The excessive consumption of animal food, especially when washed down with malt-liquors and wines, is an influential factor. Garrod first demonstrated the important fact that lead-poisoning manifests itself, in a certain proportion of cases, by paroxysms of gout. This statement, at first received with incredulity, is now universally admitted.* The explanation is, that lead greatly lessens the excretion of uric acid, and the proof is afforded in the increased quantity of uric acid in the blood. The climate has an effect on the occurrence of the seizures, winter being the season of greatest tendency to them, and hence they are often avoided by the timely transfer to a warm winter locality.

Pathological Anatomy.—The changes in the joints are characteristic when a single joint has been affected, and once only. In such a case a part of the head of the metatarsal bone was covered with a white incrustation after thirteen years (Garrod). The whole articular surface of the affected joint attacked is, in severe cases, covered with a whitish deposit, to the synovial fringes. First, a transparent fluid is exuded into the substance of the cartilage; the water is absorbed, leaving the white incrustation composed of bundles of acicular crystals radiating from a center. This material is urate of soda. Most of the articulations are, in old and severe cases, more or less affected, but the tarsus and carpus and the surfaces of the metatarsal and metacarpal bones and some of the phalanges are chiefly diseased. More or less urate deposits have been found in the bone itself. The presence of this material excites ordinary inflammation, and hence the thickening and deformity observed about the diseased joints are partly due to the products of inflammation, mixed with the chalk-like accretions of urate of soda. The blood also contains urate of soda, and in the perspiration uric acid is frequently present, and also is in excess in the fluids transuded into the pericardium and peritoneum. During the gouty paroxysm the blood is said to contain an abnormal quantity of fibrin. The most important of the changes in internal organs is that disease of the kidney known as the "gouty kidney." Crystals of urate of soda are deposited in the tubules and inter-tubular tissues, and may be seen by the naked eye as white lines. The kidneys are small, granular, and fibrous. In the vascular system, atheromatous changes of the senile type are precipitated by attacks of gout.

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* Wilks, Dr. Samuel, "Guy's Hospital Reports," 1869–'70, p. 40.

Symptoms.—ACUTE GOUT.—Gout is not always manifested by the same signs and symptoms: it may be acute, chronic, or irregular. The paroxysm of acute gout may or may not be preceded by prodromic symptoms. In many patients certain symptoms appear invariably, and announce the approaching attack. These preliminary symptoms may consist of gastric disorder—as headache, nausea, a coated tongue, constipation, a muddy skin, a yellow conjunctiva; of nervous disturbance—as restlessness, wakefulness, despondency, irritability, peevishness, or exhilaration, and high spirits, etc.; or they may experience a more or less febrile condition, as shivering, rise of temperature, and sweating. In many cases any indications of the approaching tempest are wanting. The patient is awakened out of a sound sleep about 2 A. M., or between 12 M. and 5 A. M., with a sense of uneasiness rapidly growing into acute pain in the ball of the great-toe, if a recent case. The part the seat of pain is red, hot, swollen, and so exquisitely sensitive that the lightest touch, the weight of the bedclothing, the jar of one walking over the floor, can not be borne. The veins of the foot are swollen. Now and then the muscles of the leg start with sudden spasms, and a hot pain pierces the joint. No position gives relief. If the foot be placed on the floor the veins swell still more, the joint becomes deep red, almost purple, and the pain becomes agonizing, so that the patient gladly foregoes any attempt to walk. As a rule, a feverish state develops; some chilliness is first experienced, then the temperature rises, the pulse quickens, there are thirst and a coated tongue. The urine voided during the paroxysm is dense, deep red, acid, and deposits copiously the brick-dust sediment. After several hours of severe suffering, and in the early morning, the pain abates, the skin is covered with a warm prespiration, and a general sense of relief is experienced. If, now, the foot is kept elevated and at rest, and all excitement avoided, the relief continues through the day; the joint is less red, less swollen, and less tender; but when evening approaches sharp pains again fly through the joint, the swelling rises again, and another night of agony is passed. The same experience may be repeated for several days and nights longer—exacerbations at night, comparative ease by day. If no treatment of any kind is instituted, the case may continue in this way for a week, for ten days, even for two weeks, but the usual duration under the present treatment is but four or five days. When the joint and surrounding tissue are much swollen, the pain becomes less severe; but toward the end of the paroxysm the swelling subsides, the redness also, and desquamation of the epidermis is apt to take place in fine scales, and sometimes in large flakes. The swelling veins collapse, but when the foot is first placed on the floor they quickly fill, and the whole member feels sore, and tingles, and is painful from a fine prickling. The ankle and foot are stiff and awkward for many days. The system is much depressed by an attack of acute gout, the body-weight is lessened, the lines deepen in the face. When the attack is over, the ravages committed by it are quickly repaired, and a feeling of well-being, often of exhilaration, takes the place of the hebetude of mind, and the bodily distress, or other disagreeable sensations which preceded the outbreak. The patient may continue free from gouty paroxysms for two or three years, but he is usually visited again in about a year. The same joint may be attacked as before, which is more frequently the left metatarso-phalangeal joint of the great-toe, but this seizure may be concerned with the right, or both. A similar interval may elapse before the next seizure, when the inflammation may be in the same joints as in the previous paroxysms, or may extend to the other articulations of the foot, and to the ankle. In the further progress of the case other joints are affected—those of the upper extremity, the hip, the knee, etc.—and the attacks come nearer together, until ultimately they may be expected at any time. As the paroxysms increase in number, they decline in severity, but grow longer in duration. The skin does not recover, but remains red and livid, while the veins become varicose. Meanwhile, the systemic condition tends to permanence, and the general as well as local symptoms persist.

Chronic Gout.—The distinction between acute and chronic gout consists in the wider diffusion of the articular troubles, their less pronounced character, and the preponderance of the constitutional state, in the latter or chronic form of the malady. The affections of the digestive organs, which precede the paroxysms, and are present in less degree at all times, consist of acidity, flatulence, pain about the epigastrium and through the hepatic region, distress after eating, hæmorrhoids, constipation alternating with diarrhœa, a coated tongue, and fetid and heavy breath. Sometimes the paroxysms are preceded by various nervous symptoms—especially by feelings of depression, irritability, twitching of the muscles, cramps in the legs, palpitation, and occasionally intermittence of the heart-beat. The paroxysms occur at any time, but they develop slowly, and there are less pronounced local and general symptoms, and they do not have the critical character, nor produce the relief, of the acute seizures. The deposits about the joints increase with the duration of the case; and the joints become hard, knobby, and are often much distorted. These deposits or *tophi* (chalk-stones) form not only about the joints proper, but in the tendons and bursa, producing deformity and seriously impairing the functions of the articulations. Among other places, these tophaceous deposits form on the helix of the ear.

Course, Duration, and Termination.—Gout is a very chronic disease, for, although there is an *acute gout*, this form is merely an exacerbation of the chronic disease. The first paroxysms are separated by long intervals, but after some years the *chronic gout* is established. This continues with varying fortunes for several years. The complications which increase the gravity of the disease are numerous. The chalk-stones seem at first to be important only as they deform joints and impair functions, but they are foreign bodies, excite inflammation and ulcerations which show no disposition to heal, but continue to discharge, and if numerous may wear out the strength and cause death by exhaustion. The changes in the kidneys ultimately become highly influential factors in the morbid complexus. These organs separate less and less excrementitious matter ; the urine is pale, of low specific gravity, and contains albumen. The changes in the kidneys may be the main causes of the cerebral symptoms which occur toward the end, and of the cerebral hæmorrhage with which so many gouty subjects are carried off. During the course of chronic gout, various troubles arise in internal organs, and are styled *gouty*. "Gout in the stomach," "gout in the head," are popular phrases, which indicate the general belief that gout abandons the joints to attack internal organs. This notion was also represented in the technical phrase "retrocedent gout." That such a retrocession, or metastasis, does actually occur, is no longer maintained. Important changes of structure take place in internal organs, as a result of chronic gout, and hence, indirectly, gout may be responsible for various diseases. "Gouty kidney," as it is called, and the serious result of the change have been already referred to. Atheromatous and calcareous degeneration of the vessels leads to attacks of angina pectoris (gout in the heart, in popular language) and to cerebral hæmorrhage (gout in the head). The changes in the composition of the blood, which belong to gout, are fruitful causes of acute inflammations, as pneumonia, pleuritis, etc. The mode in which cases of gout may ultimately terminate is indicated in these observations on the changes wrought by the disease. When the lesions of chronic gout are established, we must take a hopeless view of the situation. When the disease is inherited, although it may not have proceeded far, the probability of affording some permanent relief is less than in the acquired disease. When the first paroxysm has occurred, the prognosis will be greatly affected by the disposition of the patient and his power of self-control.

Diagnosis.—Errors of diagnosis are possible only in the case of chronic gout, and between this and *arthritis deformans*. The differentiation may, however, be readily made. Arthritis deformans occurs among the poor and ill-nourished—in women chiefly, and at or before middle life. There are no paroxysms ; it is gradual in its growth, and affects the two sides in a symmetrical manner, and is not accompanied by urate-of-soda deposits.

Treatment.—The treatment of gout is concerned with the paroxysm, with the chronic form of the disease, and with the intervals between the paroxysms. There are two methods of treating the paroxysms of gout—the expectant and the eliminant. By the expectant, the patient is put at rest, the joint is wrapped in cotton-wool, a laxative is administered, and the diet is reduced to slops. Under this method the duration of the attack is protracted, but the ultimate results are better than if more active treatment were pursued, provided the patient make such change in his mode of life as may be necessary. The suffering is so great, however, that the patient is usually clamorous for relief, and hence more active measures are necessary. There are but two remedies which exert a really curative influence on gout—colchicum and salicylic acid. Colchicum has been used for many years, and has demonstrated its power to alleviate the pain and shorten the duration of the acute attacks. The active principle, *colchicine*, is preferable to the crude drug. It may be given advantageously with quinine, morphine, and compound extract of colocynth. The wine and tincture may also be employed. In the various prescriptions for gout, besides colchicum there are usually an alkali, a potash-salt, and a purgative, colocynth. The object is to secure elimination of the urate of soda and prevent its deposition. Salicylates have recently been employed with great success to relieve the gouty attack. They may not be given when the stomach is very irritable, or in atonic gout, but, in the usual acute gout in a vigorous subject, the relief afforded is surely remarkable. If the stomach is very irritable, effervescing salines—the common effervescing, or the Sedlitz-powders if there be constipation—are useful by promoting elimination by the various organs of excretion. If the pain is very severe, morphine, hypodermatically, will afford prompt relief, but remedies of this kind must be used sparingly because of their effect in stopping elimination. Local treatment is of doubtful utility. Leeches applied in the neighborhood are of real service if there is much swelling, the patient robust, and the attack recent. Blisters in the neighborhood of the joint are always safe, are useful as regards the subsequent course of the case, and afford much immediate relief. Besides these measures, it is necessary only to support the foot at a considerable elevation, maintain rest, and cover the painful joint with some cotton. Excessive warmth and much covering are hurtful. A man who has suffered an attack of gout should at once change his mode of living. As to drop from an abundant and rich diet to a poor and spare diet involves much risk, the change should be made gradually. The diet of a gouty subject should consist chiefly of vegetables and fruit; he should take fresh meat once a day; coffee and tea should be given up, and skimmed milk substituted; eggs are also injurious, and all dishes into which eggs enter; pastry, cakes, hot bread, sweetmeats, spices, and condiments, are to be avoided, while oysters, fish, soups, may be eaten. Next to careful regulation of the diet, exercise is most important. Walking, riding, rowing, but especially walking, should be carried out systematically, and, when inclement weather prevents exercise without, it should be done in-doors. If no other mode of exercise is possible, passive movements, massage, and faradization of the muscles, can be conducted in bed if need be. Cold bathing is objectionable. The patient should wear flannel, and migrate from a cold winter climate to a warm one if his means permit. Certain kinds of waters are serviceable: in this country, Saratoga, especially the Vichy spring, the alkaline waters of Wisconsin, and of St. Catharine's, Canada, the Warm Springs of Virginia, and the Hot Springs of Arkansas; abroad, Vichy, Carlsbad, Wiesbaden, Homburg, etc. Elimination may be maintained by drinking freely of ordinary drinking-water. Much of the efficacy of alkaline waters is due to the quantity of fluid swallowed. Excellent results are obtained from the use of the lithia salts in chronic gout. These preparations promote the excretion of uric acid, and apparently the solution of the deposited urate of soda. The interval between the attacks is lengthened, and the attacks are less violent and of shorter duration, when the citrate of lithia has been given for some time. In atonic gout a modified course must be pursued. With the potash and lithia salts must be combined quinine and iron; the food must be nourishing without being abnormally stimulating, and massage and faradism perform the part of active exercise.

ARTHRITIS DEFORMANS.

Definition.—By *arthritis deformans* is meant a chronic inflammation of the joints, without fever and without suppuration, progressive, and causing nearly symmetrical enlargement and deformity of various articulations. It is called *rheumatoid arthritis* by Garrod, and *rheumatic arthritis* and *rheumatic gout* by various authors. As the supposed rheumatic character of the disease is more than doubtful, the term employed by the German writers—*arthritis deformans*—is preferable, because no theory is coupled with it.

Causes.—Arthritis deformans does not appear to be propagated by hereditary tendency. It is more especially a disease of women than of men, and is apparently associated with disorders of the menstrual function, particularly at the climacteric period. Cases do occur among men, and sometimes they are exceptionally severe. Poverty and bad hygiene, exposure and hard work, with inadequate food, prolonged lactation and frequent pregnancies, are among the most influential causes. Garrod holds that it may have its origin in the tubercular diathesis. It is usually regarded as a disease of advanced life, but cases occur from the period of puberty on. Moral causes are very influential in its production—for the disease has repeatedly followed grief, anxiety, and moral depression. As various changes in the joints are produced by certain troubles of the spinal cord, a state of the nerve-centers is invoked to account for this disease. Joints that are injured, as the ball of the great-toe by a tight shoe, are the first to undergo the change.

Pathological Anatomy.—At an early period there are seen only the changes of inflammation—hyperæmia of the synovial membrane and an increased amount of fluid in the joint. After absorption of fluid has occurred, the capsule of the joint is found to be thickened, and the ligaments are elongated, thus permitting ready dislocation. The cartilages are absorbed, and the bones rubbing together are polished and hard, like ivory, a condition which is called "eburnation." The articular extremities become thickened and broader, and are flattened out, their margins projecting, and studded with irregularly rounded bony outgrowths. The fluid contents of the affected joints consist of a much altered synovial fluid, especially rich in mucin, and containing cholesterin and lecithin (Hoppe-Seyler[*]). In occasional cases the capsule of the joint is partly or wholly ossified. Not only the joints, but the adjacent tendons and their sheaths and the bursæ, become ossified, and the muscles waste and undergo fatty degeneration.

Symptoms.—Slow enlargement of a joint that is exposed to injury, as the wrist in a laundress, the thimble-finger in a seamstress, or, after a more or less prolonged period of trouble and anxiety, the general health being reduced by nursing, the knee or some other joint becomes painful and swells. The first attempt may subside, and presently the same joint or another may undergo the same process, but a subsidence no longer takes place, and the joint remains swollen. In a short time other joints are attacked. In other cases the first symptom experienced is pain in the articulations, which subsequently become swollen. The joint is sensitive to atmospherical changes, and feels sore when flexed or extended. Acute pains extend along the nerves in the neighborhood. Thus, if the changes have begun in the hip, the pain is felt in the sciatic nerve. After the pain has continued for some time, the joints are observed to be enlarging. The fingers and toes, knees and wrists, are affected in the more youthful subjects, while, in the senile, the hips, spine, and shoulders are more especially visited. When the deposits about the joints have attained a certain magnitude, their mobility is lessened. After a more or less prolonged rest the parts become rigid, and motion is difficult until the persistent use of the members limbers them again. The osseous deposits about the joints and tendons at length reach such a stage of development that the affected joints have a very limited range of movement. The thickened joints are not red, but pale, and, although painful, are not tender. The changes in the articulating surfaces and the relaxation of the tendons lead to subluxations. When the articular cartilages are removed, and the ends of the bones rub together, a grating is produced that is felt by the patient and through the soft parts. This crepitant sound may also be due to the movements of the tendons through their partially ossified sheaths, or by the collision of the osseous masses which form about the various articulations. The hands are peculiarly prone to take on this deformity. The heads of the metacarpal bones and the phalanges are distorted by large nodules. "The metacarpo-phalangeal articulations of the fingers are flexed, the first phalangeal extended, causing the second phalanx to be thrown backward, and the second phalangeal joint is also flexed. The phalangeal joint of the thumb is usually extended or bent backward" (Garrod). When the larger joints of the lower extremities are affected, especially the hip, the gait has a characteristic halt and limp. The spread of the arthritis through the articulations is symmetrical, or nearly so.* The muscles of the limbs waste, the subcutaneous fat disappears, and hence the members have a wasted appearance, which recalls the myopathies of spinal origin. When the vertebræ are affected, anchylosis takes place, reducing the flexible spinal column to the rigidity of an iron bar. Various ill results follow. If the cervical vertebræ are anchylosed, the patient's head is kept erect and rigid without power of bending or turning; if the dorsal and lumbar vertebræ are anchylosed, the body is twisted and immovable. In the worst cases, finally, all the joints are spoiled, are fixed in bony anchylosis, and motion is no longer possible.

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[*] Virchow's "Archiv," Band lv, s. 252.

Course, Duration, and Termination.—Arthritis deformans is one of the most chronic of diseases, continuing on its course for ten, twenty, even thirty years, or longer. It is a progressive disease, and does not cease or get well spontaneously, yet it sometimes remains stationary for months and years at a time. Although of itself not affecting the constitution in a marked way, and sometimes not at all impairing the general health, in other cases life is rendered intolerable and the strength is exhausted by suffering and loss of sleep. Most obstinate sciatica may attend on the disease in the hip, and neuralgia, contractures, paralyses, etc., may be caused by the osseous deposits along the spine. Otherwise the disease continues through life, not apparently abridging it.

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* Hutchison, "Transactions of the Pathological Society," vol. xxiii, p. 194.

Treatment.—The only remedies which have appeared to do any good are iodine and galvanism. The compound solution is an eligible form, which we may give in the dose of five minims, three times a day. Iodine-ointment may be carefully rubbed into the affected joints. The oleate of mercury and morphine may also be painted over (not rubbed in) the joint, and along the course of painful nerves. Galvanic currents should be transmitted through the cervical sympathetic, and be applied also to the affected parts, the principal nerve-trunks being included in the circuit. As many as forty to sixty cups should be used, and large, well-moistened sponge electrodes should be applied. Warm baths, massage, passive motion, and faradization of the muscles, are among the very useful expedients to be employed in these cases. Undoubtedly good results have been obtained from the use of arsenic, if given early in the disease. If anæmia exist, as is so often the case, iron is necessary. If the nutrition is low, cod-liver oil and the hypophosphites may be given with advantage.

DIABETES MELLITUS.

Definition.—*Diabetes* is a chronic disease characterized by the constant presence of grape-sugar in the urine, by an increased urinary discharge, and by progressive wasting of the body. The occasional and temporary presence of sugar in the urine does not constitute *diabetes mellitus*, although it may precede the fully developed disease. *Diabetes insipidus* is a malady in which the urinary water is largely increased in amount.

Causes.—Climate exerts a certain influence in the causation of diabetes, but the influence is capricious and there are no obvious reasons for the greater prevalence of this disease in one locality than in another. Race seems, in respect to one people at least, to be concerned—the Jews, who are apparently more frequently the victims of diabetes than the Christians. It is distinctly hereditary, and, although this fact has not been properly appreciated heretofore, the examples of hereditary transmission are becoming so numerous that this will hereafter occupy a high position in the etiology of the disease (Senator). Diabetes is more common in males—three to one, according to Brunton,* who bases his statement on the statistics of eight German and French authors. But this proportion does not hold good for children, with whom females are more given to the disease (Durand-Fardel, Senator), and this is the experience of the author. Diabetes occurs at all ages, but is most frequent in middle life—from thirty to forty for males, and from twenty to thirty for females. There are two types of subjects addicted to the disease, the obese and the thin, and they represent two kinds of causes. The obese are addicted to the pleasures of the table, suffer from a certain kind of indigestion, and are given to sedentary habits. In the thin and nervous subject the disease comes on after some excitement, chagrin, business failure, or other cause of cerebral disturbance. Among the exciting causes must be placed mechanical shock, concussion of the whole body, or of the brain and spinal cord, blows upon the hepatic and renal regions, etc. Mental shocks, profound moral impressions, especially anxiety and chagrin, are, in the author's experience, very common causes in the class of subjects mentioned above; but, in the obese class, errors of diet, the consumption of a large proportion of farinaceous food and of malt-liquors are chiefly responsible. The occurrence of acid indigestion and the probable formation of lactic acid in the intestinal canal (the duodenum) are elements to be considered in this connection. To these exciting causes must be added exposure to cold and wet while the body is heated, sexual excesses, extreme fatigue, etc.

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* Reynolds's "System of Medicine," article "Diabetes."

Pathological Anatomy.—There are two groups of morbid alterations: those which stand in an apparently causative relation to the disease; those induced by it. In the intestinal canal the changes consist in a proliferation of the epithelial layer of the mucosa throughout the whole tract, in hyperæmia and thickening of the mucous membrane, and also sometimes of the muscular layer. The muscular tissue of the heart is relaxed and fatty, and the vessels, especially the median and small-sized vessels, are atheromatous, the atheroma being more decided in the cerebral vessels at the base than elsewhere in the body. The blood is altered by a great increase in the amount of fat in the serum, which may even have a milky appearance from this cause. Atrophy, cystic degeneration, and, according to some, hypertrophy of the pancreas, have been observed, but atrophy occurs in one half of the cases —a fact of great pathological importance. More significant changes occur in the liver, but these are by no means constant, for the liver has sometimes appeared to be quite normal. In twenty-seven cases examined by Dickinson the liver was healthy in six. In Seegen's cases at the Vienna Hospital, thirty in number, fifteen presented obvious changes in the liver. In some cases which have been reported, the liver was enormously enlarged. The most constant changes consist in an active hyperæmia, generally diffused, the acini appearing as well-defined rose-colored spots surrounded by distended capillaries; in enlargement of the hepatic cells with rounding of their contour, and occasionally in hypertrophic enlargement of the connective tissue of the organ. The kidneys are in an obvious pathological condition in more than one half of the cases—usually enlarged and decidedly hyperæmic, without being otherwise altered. More or less fatty change ensues in some instances, the infiltration of fat occurring in the cortical portion chiefly, giving to it a pale-yellowish appearance, and increasing its thickness. This fatty infiltration is no doubt due to the persistent hyperæmia. Various morbid changes have been discovered in the brain and spinal cord, but they are by no means uniform in position or character. Hyperæmia, dilatation of the perivascular lymph-spaces, remains of old extravasations, pigmentations, fatty degeneration of cells, tumors, etc., have been found in various parts of the brain, cord, medulla, pons, etc. Important lesions, also, have been made out in the semi-lunar ganglion, solar plexus, and splanchnic nerves; they have been seen much enlarged, thickened, and of almost cartilaginous hardness. These changes appear to be the cause of the extraordinary wasting of the pancreas which so often takes place. The lungs are frequently far advanced in phthisis. In only two of twenty-seven diabetics under the observation of Dr. Dickinson were the lungs free from the various alterations of phthisis at some stage of its development. The body at death is extremely emaciated. Remains of ulcers, abscesses, and gangrene sloughs are to be seen in the skin of various parts. The muscular tissue is dry, anæmic, relaxed, and its color pale, but it is sometimes of a reddish-brown.

Symptoms.—There are two distinct types of subjects who are affected by diabetes: the obese and phlegmatic; the thin and nervous. The onset and the behavior of these two varieties are very different. A recognition of the peculiarities of each is necessary to a proper comprehension of the malady. In the obese subjects the onset of the disease is gradual; they experience, for a long time previous to the beginning of the malady, disorders of digestion; they suffer from acidity, pyrosis, and a sense of epigastric weight and uneasiness. Notwithstanding the obvious derangement of the digestion, they have a keen, almost an insatiable appetite, and a strong thirst, and they constantly increase in weight up to a certain point. They pass, at this period, an excessive amount of water, and the urine occasionally contains sugar, but not constantly by any means. They are troubled with boils or carbuncles, and often have hard, inflammatory swellings, which slowly suppurate, and discharge with a considerable slough, leaving an indolent ulcer behind which shows but little tendency to heal. In the thin, nervous type, the opposite conditions obtain. These subjects are nervous, suffer from attacks of neuralgia, and are rather hypochondriacal. With them, digestion is never active; they are rather constipated, and the functions of the gastro-intestinal canal are as a rule performed with a certain feebleness, without there being any pronounced derangement. The disease usually comes on abruptly. There may have been headache, neuralgia, or mental despondency, but these symptoms have no necessary connection with diabetes. After some business troubles, anxiety, grief, or other moral cause, it is observed that there is an unusual urinary discharge, that the strength is exhausted by the least effort, and that a sense of fatigue is constant. When the disease has really begun, there are present constant thirst, dryness of the mouth and tongue, an unusual appetite, and frequent discharge of urine, in large amount at a time. In other cases the vision is impaired, and the diagnosis is made by the oculist, to whom the patient has repaired for advice about his eyes. The thirst is excessive, and the amount of water and of other fluids drunk is enormous ; the appetite becomes voracious, insatiable, and the individual, who previously had been rather indifferent to food, now gloats over the viands placed before him, and thinks only of satisfying his appetite. A frequent desire to micturate comes on with the thirst, the patient is disturbed repeatedly at night, and in the morning the vessel contains a much larger quantity than usual. The aggregate amount passed in twenty-four hours may reach 80 to 100 ounces or more; it is acid in reaction, and has a specific gravity of 1020 to 1040, even 1050. The bowels are confined, the fæces hard, and voided with difficulty. The saliva is acid. The tongue is pasty, deeply fissured, sometimes blackish, dry, and hard. The gums may be soft and spongy, the teeth loose and apparently elongated, because of the retraction of the gums. The breath has a peculiar mawkish, disagreeable odor, likened to that of new hay or of new apples. The skin becomes dry and rough and is attacked by herpes or eczema, and, when emaciation proceeds to a considerable extent, is wrinkled and inelastic. The eyelids may be swollen. In one of the author's cases, ptosis appeared with the first symptoms. Headache, vertigo, double vision, neuralgia, wakefulness, deep dejection of mind, abnormal sensations in the skin, formication, are nervous symptoms, especially apt to occur in the thin, nervous type of subjects. The sexual appetite early declines, and is soon wholly absent, the erections ceasing permanently. Itching at the orifice of the urethra is an early symptom in both sexes, but especially in women. The itching may extend from the meatus to the vulva generally, and produce intolerable torment. Whenever this symptom occurs in obese women, the urine should always be examined. The prepuce and the vulva, also, are excoriated by the passage of the saccharine urine so frequently. Such are the symptoms of the disease in its process of development. It is necessary now to indicate with somewhat more detail the chief features of the malady at its maximum.

The remarkable increase in the urinary discharge is the most striking phenomenon. We have already mentioned eighty and one hundred ounces as a not unusual quantity, but these figures have been largely exceeded in some cases, e. g., Bence Jones, who reports a case passing seven gallons. On the other hand, the urine may not be increased above the normal, or may fall below it. Toward the end there may be a notable decline in the quantity of urine, and this fatal symptom may be entirely misconceived. The amount of urine discharged stands in a nearly constant ratio to the amount of water drunk. The apparent exceptions to this are cases of patients unable to swallow much fluid, the surplus over that taken into the body being formed by the oxidation of the hydrogen, or supplied from the water stored up in the tissues. The urine of diabetes is clear, of a faint greenish tinge, and is free from sediment. If it stand for some time in a warm place, it is covered with the *Torula cerevisiæ*, or yeast-fungus. The urine is acid in reaction. The specific gravity, as already stated, ranges from 1020 to 1050, but it may contain sugar, and yet fall below normal. The variety of sugar present in the urine is *grape-sugar* and not *cane-sugar*, the variety in domestic use. The former differs from the latter in the readiness with which it ferments, in turning the plane of polarized light to the right, and in its source, the grape-sugar of commerce being obtained from starch by the action of sulphuric acid. Grape-sugar is also less sweet than cane-sugar, and is harder in texture. The actual amount of grape-sugar present in urine ranges from a mere trace to ten, even fourteen per cent. Dickinson reports an extraordinary case of a man who excreted in twenty-four hours fifty ounces of sugar. The quantity of sugar stands in a certain ratio to the amount of urine —the larger the flow of urine the greater the quantity of sugar voided ; and to the character of the food, for the more sugar and starch in the food the more sugar in the urine. The high specific gravity of the urine is not wholly due to the presence of sugar, but is also influenced by the quantity of urea, which may rise to a proportion two or three times greater than the normal. This increase of urea is due to the largely increased consumption of nitrogenous diet, and to the greater metamorphosis of the nitrogenous tissues. As the formation of urea is one of the hepatic functions, the increased production of this substance may be due to the heightened functional activity of the liver. Albumen is present in a proportion of cases not definitely settled. It may be due to the increased blood-pressure. Irritation of a spot on the floor of the fourth ventricle causes albumen to appear in the urine, as irritation of another spot below causes an excretion of sugar. *Inosite*, or muscle-sugar, has taken the place of grape-sugar in some rare cases. *Acetone* has also appeared in the urine in a few cases. Various affections of the special senses occur during the course of diabetes. Ptosis has been mentioned. Amblyopia, paralyses of accommodation, and amaurosis, also occur. The most striking phenomenon connected with vision is the occurrence of cataract, which is encountered in the proportion of one in twelve to one in forty-five cases. The cataract is of the soft variety, and both eyes are usually attacked, that in the right eye developing more rapidly. The formation of cataract is simply a failure of the nutrition of the lens in consequence of the state of the blood. Owing to the same cause, boils and carbuncles appear among the prodromic symptoms and also at the maximum of the disease. Carbuncles may indeed be the cause of death. Gangrene of the skin, and gangrene of a toe, foot, or leg, may also occur. The great loss of material continually going on must necessarily cause wasting, emaciation, and a sense of fatigue. A rapid accumulation of flesh—of adipose—takes place in the obese subjects of diabetes when the disease begins, for then the retrograde changes through the channels of excretion are not so active as the source of supply. But presently the waste exceeds the supply, and then a rapid loss of weight is observed. Patients going through this process present a very characteristic appearance: they have an old look, and may be much wrinkled; the skin is rough, cracking at the ends of the fingers, and the countenance wears an anxious expression. The lips are pallid, the mouth dry, the tongue dry and hard, and constant smacking of the lips and sucking of the tongue, in the vain effort to moisten the parts, are characteristic of diabetics. As the nutritive functions are so depressed, it is not surprising that the temperature of the body should remain below the normal. It has been found as low as 93·2° by Dickinson. Foster* has pointed out the very curious fact that the temperature of the fluids drunk exercises an influence on the temperature of the diabetic patient. His figures show that, when all fluids drunk were warm, the temperature of the axilla was one degree higher than when the fluids were cold.

Course, Duration, and Termination.—In the obese type the prodromes may continue over several months, even years. There may be occasional glycosuria, of variable duration, occur several times, before the persistent presence of sugar constitutes the case one of diabetes. On the other hand, in the nervous type, the preliminary symptoms are of brief duration. So long as the appetite and digestion are equal to the supply of all the material excreted, the patient holds his own. When, however, the loss is in excess, the decline is rapid. The cases vary greatly in the rate of progress. Those diabetics, in whom the proper regulation of the diet causes a disappearance of all the symptoms, apparently recover, and the duration may therefore be much prolonged, but they ultimately succumb, because they at length reach a period when they can no longer prevent the formation of sugar. Those cases proceed rapidly in whom the changes of diet make but little difference in the formation and excretion of sugar. The average duration is about two to three years. Under proper management favorable cases, not curable, may continue for many years, the patient meanwhile enjoying good health. Age has much to do with the rate of decline. The disease makes very rapid progress in children. The most acute cases terminate in a few weeks. The prognosis is most unfavorable as regards cure, and gloomy in respect to retardation. The reports of recovery are discredited by those of greatest experience. While cures may not be hoped for, in a considerable proportion of cases decided amelioration may be accomplished and a retardation of great length effected in a small number. Much depends on the influence of the changed diet over the excretion of sugar; for, if, on a nitrogenous diet, the sugar disappears from the urine, the case wears a decidedly more hopeful aspect than if the formation of sugar continues despite the change. In the further progress of the case, the condition of the patient will depend largely on the behavior of the digestive organs as confined to an animal diet. If he can not persist in this diet, and his digestive organs are upset by the nitrogenous food, a prompt development of the worst symptoms will ensue. The mode of dying is various. Death by exhaustion is not common. In some cases, apparently doing well, the most serious symptoms, known as *diabetic coma,* suddenly appear. Great restlessness, præcordial uneasiness, and pain are quickly manifest; somnolence, with general agitation, loud cries and groans, and then a deepening coma, come on, during which the pulse grows weaker, the respirations more and more shallow, the temperature lower and lower, and soon the patient expires in a condition of profound insensibility. These symptoms of such formidable character, and arising suddenly, wear an aspect of poisoning, very like that caused by acetone in animals. As this substance may be produced in the blood by the decomposition of the diacetate of ethyl—a product of the reactions of grape-sugar—it is assumed that diabetic coma is an *acetonæmia.* The most frequent cause of death is phthisis. This may develop very slowly and escape detection until far advanced, or it may proceed very rapidly and with pronounced symptoms from the beginning. Gangrene of the lung rarely occurs, and it presents the remarkable peculiarity that the sputa are without odor.

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* "Clinical Medicine," by B. Foster, M. D., Philadelphia edition, p. 264.

Diagnosis.—Diabetes exists only when sugar is permanently present in the urine. In *diabetes insipidus* there is a large flow of water, but no sugar; in glycosuria of the temporary kind sugar is only occasionally present. It is not the quantity, but the persistence of the sugar, which constitutes diabetes. As Senator well expresses it, "a saccharinity of over *two per cent.* certainly occurs in diabetes, but a lower saccharinity does not exclude diabetes." The urine of diabetes has certain physical peculiarities by which it may be recognized, but not with the certainty of chemical reactions. It is free from sediment, has the appearance of water of a very pale-greenish tint; has an acid reaction, and a specific gravity of 1025 to 1050 or higher. The importance of a high specific gravity depends on the fact that the quantity of urine is also large. The presence of sugar is the important point, and this must be determined by the applications of chemical tests:

Trommer's test is the most generally applicable. A few drops of a dilute solution of sulphate of copper are added to some urine in a test-tube, or sufficient to give to the urine a blue color, faint but yet distinct. Then the same quantity of *liquor potassæ* as there is urine is added. If sugar be present, the precipitate at first formed is redissolved, and the mixture assumes a deep-blue color. If, now, heat be applied, a yellow or orange-red precipitate of oxide of copper is thrown down. If the heat be applied to the upper part of the liquid, the vivid yellow color of the oxide of copper appears bright and distinct by comparison.

Fehling's test solution must be kept prepared, and, as it rapidly spoils by keeping, frequent renewal of the solution is necessary. This test consists of a sulphate-of-copper solution, mixed with a solution of tartrate of soda and potassa (Rochelle salt) and caustic soda. Some of this test is added to the urine in a test-tube and boiled, the reaction being the same as in Trommer's.

Fermentation Test.—Some brewer's yeast is added to the urine in a bottle and kept at a proper temperature (60° to 80° Fahr.). The bottle must be well corked and have a bent tube connected with it, so that the carbonic acid can be collected for examination.

Moore's test consists in simply boiling together equal parts of urine and *liquor potassæ*. The sugar is decomposed, and one of its products is melassic acid, which may be recognized by its odor, and which, with glucic acid, another product, converts the mixture into a brown, almost a black color. The change of color is rendered all the more evident by confining the heat to the upper portion of the mixture.

Treatment.—The most important points in the management of this disease are diet and exercise. Medicines are secondary. As the presence of sugar in the blood is the great cause of mischief, our efforts must be directed to prevent its formation. This can be accomplished only by systematic disuse of all articles of food convertible into sugar. Bread, potatoes, beans, peas, rice, carrots, turnips, parsnips, etc., and all articles containing flour, sugar, or starch, must be excluded. Greens, cabbage free of the stock and stems, lettuce, tomatoes, and spinach, may be substituted. A bread made of powdered almonds and gluten bread may also be substituted for ordinary bread. Milk may be allowed, especially buttermilk. Donkin reports cures made by confining the patient to an exclusive diet of skimmed milk—six to eight pints daily. Brunton objects to the skimmed milk because some die of inanition, although he says others do recover. All kinds of flesh, fresh or salt, fish, including oysters, eggs, gelatine, fats, almonds and nuts, except chestnuts, are allowed *ad libitum*, unless the too great consumption of animal food induce the excessive excretion of urea and uric acid. Koumiss may be taken—also light acid wines and a little spirit with meals. Water may be taken to satisfy thirst, but a large quantity of fluid at meals must prove detrimental to digestion and should not be indulged in. Walking exercise is of the highest moment. There is a strong sense of fatigue proper to the disease, and the muscles are actually unequal to much effort. Surprising results may be accomplished by active walking exercise, especially in the case of the obese diabetic. The strength is improved, and the formation and excretion of sugar are diminished. The functions of the skin should be maintained by warm clothing. It is probable that pilocarpine will prove beneficial by increasing the secretion of the sweat and saliva. Of the medicinal remedies but few have proved beneficial. Opium, and especially the alkaloid codeine,* exercises a great influence over the excretion of sugar, but unfortunately the effect is not maintained. Large doses are well borne and are required. Next to opium is arsenic, which, especially in the form of the bromide of arsenic, has proved highly useful. The alkalies are also useful, especially the phosphate of sodium, which the author has found in some cases, those of full habit especially, to exercise an apparently curative effect. The weak alkaline waters of Wisconsin (Bethesda), drunk in large quantities, seem to have a very beneficial influence. The Carlsbad water of Germany and Vichy of France have had a reputation for generations in the treatment of diabetes. Saratoga Vichy, and our native lithia waters, may be substituted for the foreign waters. The bromides have proved useful in some cases, and the best results have been obtained from the use of bromide of ammonium. The carbolate of iodine (carbolic acid ʒ j, tincture of iodine ʒ ij, given in the dose of one or two drops well diluted three times a day) has seemed to have excellent effects in some cases, and therefore deserves more extended trial. Lactic acid has proved very useful in many cases, and in the author's hands, in the form of lactophosphate of lime, has seemed to benefit the thin, nervous type of diabetics remarkably. In this class of cases the lactophosphate of lime and cod-liver oil have been even more advantageous. Lactic acid often produces rheumatism, which is an objection to its use, and may require its suspension. Foster holds that the good effects of the skimmed-milk, whey, and buttermilk cures are due to the formation of lactic acid from the lactin. Other drugs employed on theoretical considerations are ethereal solution of peroxide of hydrogen and valerian. Any good effects derived from the former are attributed by Foster to the ether. Valerian slightly influences the excretion of sugar, but has a remarkable effect on the urea, the excretion of which lessens considerably under its use. To these drugs must yet be added the fluid extract of ergot. This drug has not been of any real utility in diabetes, but has apparently cured cases of diabetes insipidus.

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* Pavy especially eulogizes the good effects of codeine.

DIABETES INSIPIDUS.

Definition.—*Diabetes insipidus* is a disease having for its chief clinical feature the passage of a very large, often an enormous, quantity of pale, watery urine, free from albumen and from sugar. It is also known as *polyuria, polydipsia*, etc.

Causes.—Occasional examples of hereditary transmission have been reported. The disease occurs at all ages, but is most frequent from twenty to forty-five. Men are more subject to the disease than women. Among the exciting causes are injuries and diseases of the brain, including concussion, tumors, exostosis, psychical impressions, etc.; exposure to draughts of cold air, the body perspiring freely; drinking freely of cold drinks; sudden variations of temperature; fatigue, convalescence from fevers, etc. According to the author's experience, the most usual cause of the disease is syphiloma of the brain.

Pathological Anatomy.—But infrequent opportunities have occurred for a study of the changes proper to this disease. Two classes of lesions have, however, been ascertained: of the brain and sympathetic ganglia; of the kidneys. In the brain, changes have been found in the fourth ventricle—inflammatory and degenerative—tumors in this region and in the cerebellum, tubercles, syphilitic tumors, etc. Degeneration of the solar plexus has been observed by Dickinson. The changes observed in the kidneys are various—sometimes the organs are enlarged and hyperæmic; sometimes the only change is dilatation of the tubules. Other lesions, which must be regarded as accidental, have been noted, as cancer of the liver, tumor of the uterus, and disease of the mesenteric glands.

Symptoms.—It is rare for the disease to begin in perfect health without any warning. The rule is, that the nervous disturbances associated with the various lesions of the brain occur. In the author's observation there were the usual symptoms of cerebral syphilis preceding the outbreak of polyuria. The large flow of urine is the growth of several weeks. When the maximum is attained the flow is prodigious, but it is by no means the same in all cases, for it varies from one to five gallons daily. The single discharges are large, because the urine is not stimulating, and can, therefore, be longer tolerated by the bladder. The specific gravity of the urine is as low as 1002, and does not go above 1007. The urine is pale, usually clear, faintly acid in reaction, and readily decomposes. The solid constituents are somewhat increased. The excretion of urea is slightly greater than that of a healthy individual consuming the same amount of animal food, and is simply due to the increased waste of the nitrogenous elements produced by the passage of so much water through the tissues. Uric acid is diminished, as might be expected, because of the increased formation of urea. The sulphates, phosphates, and chlorides, are also increased. There is no sugar present. The reported cases of diabetes insipidus with albuminous urine were, doubtless, examples of fibroid kidney.

When there ensues such a strong outflow through the kidneys, the results of the loss of so much fluid and solid material are the same as those of similar fluid discharges. There occur excessive thirst, a dry mouth, dry skin, and constipation. The increased excretion of urea explains the diminution of body-weight which takes place in this disease, notwithstanding the appetite and digestion remain at the normal. Sometimes a decided lowering of temperature is observed, but this may be due to the ingestion of a large quantity of cold drinks. Instead of dryness of the mouth, there may be ptyalism. The skin, although usually dry, as stated, may be normal, and there may be profuse perspirations.

Course, Duration, and Termination.—The onset of the disease may be preceded by the symptoms of cerebral disturbance, due to the existing lesions of the brain and sympathetic ganglia. The increase in the flow of urine and the consequent thirst may develop slowly, and be observed only when they are very pronounced. In still other cases, during convalescence from some acute malady, or after some violent mental or moral shock, or some severe blow on or concussion of the brain, the disease begins abruptly. In most cases the disease is rather an inconvenience, owing to the frequent calls to micturate and the incessant thirst, than a dangerous malady. Death has resulted in as short a time as four months, but here the fatal result was due rather to associated lesions than to diabetes insipidus. Death may result from the disease, the continual loss of material leading to fatal exhaustion, but it is usually due to some intercurrent disease or cerebral lesion. Although death is rarely due to the disease, the prognosis is not favorable as to cure, unless caused by syphilitic disease of the brain.

Diagnosis.—Those temporary states in which a large quantity of urine is voided for a few days are all separated from diabetes insipidus by the lack of permanence. From diabetes mellitus it is differentiated by the specific gravity of the urine and by the presence of sugar. Diabetes insipidus is most apt to be confounded with fibroid kidney, for in the latter disease a quantity of pale, watery urine is passed, but it contains more or less albumen and hyaline and waxy casts, which are not present in the former.

Treatment.—Several remedies have been of real service; many others of no value. The iodides and mercury have quickly cured cases of syphilitic origin. Jaborandi has been successful in Laycock's hands, ergotin in those of Ringer and Da Costa. Valerian has been beneficial but not curative (Trousseau). Galvanism has certainly been of signal service in several cases, applied by one electrode to the neck below the occiput and the other to the hypochondriac regions in turn. From the point of view of the experience thus far gained, the following plan seems most promising: A course of iodide of potassium should be at first administered. The disease not yielding, galvanism should be used, and pilocarpine and ergotin should be tried successively if the first fails. Warm clothing should be worn, and a warm winter climate should be selected if practicable.

ALCOHOLISM.

Definition.—By the term *alcoholism* is meant the physical and mental changes induced by alcohol. The effects of a large quantity taken in a short time are known as *acute alcoholism*, and the term *chronic alcoholism* is applied to that state which is the product of the long-continued action of considerable quantities of the poison. *Mania a potu* is a delirium caused by the action of alcohol in large quantity in certain susceptible subjects: it is an acute alcoholic delirium. *Delirium tremens* is a delirium with trembling, occurring in the course of chronic alcoholism, and is often induced by bodily conditions, as stomach derangement, which prevent the introduction of the accustomed stimulant. Usually, however, these terms are applied indiscriminately to both kinds of cases.

Pathological Anatomy.—ACUTE ALCOHOLISM.—The mucous membrane of the stomach is more or less red—often vividly so—from hyperæmia. The redness is not usually universal in the stomach, but in patches, the mucous membrane about the cardia being chiefly affected. There are, also, cloudy swelling and more or less detachment of the epithelium. Here and there are occasionally ecchymoses, and still more rarely ulcerations, which form in the lower part of the œsophagus and in the stomach. A more or less intense hyperæmia, also, is evident in the mucous membrane of the trachea and bronchi. The lungs present the changes due to hypostasis, and less frequently of hepatization. Similar conditions are found in the brain. The membranes are more or less deeply injected, the *puncta vasculosa* more numerous than in the normal, and an œdematous state of the pia, of the perivascular lymph-spaces, and of the brain-substance itself, exists. It is the condition, indeed, known to morbid anatomists as a "wet brain."

CHRONIC ALCOHOLISM.—There are but few organs and tissues not in some way changed in this condition of the system. The mucous membrane of the intestinal tract presents the usual evidences of chronic catarrh. The connective tissue, especially around the tubular glands, undergoes hyperplasia, and, in contracting, subsequently encroaches on the caliber of these glands, which in consequence atrophy and degenerate. The mucous membrane is at first thickened, in consequence of the overgrowth of connective tissue, but the subsequent contraction leads to atrophic changes, to shrinking. Extravasations of blood now and then occur, and, in the transformations which ensue, appear finally as patches of pigment, rather thickly disseminated throughout the organ. Very frequently superficial ulcers—erosions of the follicles—take place. The sub-mucous connective tissue always undergoes hypertrophy. The cæcum next to the stomach is the principal seat of these changes. But more important even are the changes taking place in the liver. This organ may be found enlarged, with its connective tissue in a state of active hyperplasia, or shrunken, nodulated, and hard, in the condition of sclerosis, or it may be more or less advanced in fatty degeneration. These changes have been already described under their appropriate heads, and need, therefore, only to be mentioned here. Hyperplasia of the connective tissue of the kidneys and subsequent contraction also take place, forming the condition of chronic interstitial nephritis ; but this is not so frequent a change as the corresponding disease of the liver. The constant presence of alcohol in the blood alters its constitution in that it contains an excess of fatty matters, the minute vessels undergo atrophic changes also, and the functions of the sympathetic are depressed, so that local congestions are apt to ensue, as in the lungs. The walls of the veins sometimes undergo great thickening, encroaching on the lumen of the vessels. The muscular tissue of the heart may undergo fatty degeneration,* and in the circulatory system there ensue, earlier and more advanced, the senile changes of later years. Not less important are alterations in the structure of nervous tissue. The neuroglia of the brain undergoes hyperplasia, the ganglion-cells atrophy, the perivascular lymph-spaces are dilated, the vessels are atheromatous. The final result is, that the brain-substance is firmer, shrunken, and the vacant spaces are filled with fluid. These changes are not equally advanced in all cases, nor do they always exist together, but in old subjects of chronic alcoholism they will be found in various degrees and stages of development. The membranes are also affected in various ways and to different degrees—opacities, thickening, exudations, etc., being by no means uncommon. Pachymeningitis, with hæmatoma, is now understood to have its origin in chronic alcoholism. This condition may, indeed, be induced experimentally in animals—in dogs—by feeding them a long time alcohol with their food.† Similar changes occur in the neuroglia, in the vessels, and in the ganglion-cells of the spinal cord.

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* Dr. A. Baer, "Der Alcoholismus," etc., Berlin, 1878, p. 67.

† Kreminansky, "Ueber die Pachymen. int. hem. bei Menschen und Hunden," Band xlii, pp. 129, 321, Virchow's "Archiv."

Symptoms.—ACUTE ALCOHOLISM.—The condition of alcoholic intoxication is too familiar to require description here. The symptoms of profound intoxication, short of lethal, however, are important, if for no other reason, for the intricate diagnostic points involved. When a large quantity of some alcoholic fluid is taken, the stages of excitement and of rambling, with incoherent muttering, are soon passed; the power of voluntary control is lost, and complete muscular resolution takes place, and the patient lies unconscious, relaxed; urine and fæces discharging involuntarily. The face is bloated, congested; the lips swollen and purplish in color; the veins of the face and neck distended; the conjunctivæ injected, the pupil contracted, no reflex movements excited by touching the cornea or titillating the fauces; the breathing slow, stertorous, and shallow, with puffing expiration, and the pulse feeble and slow. Such is the condition in severe cases of alcoholic intoxication. A man so affected is said to be "*dead drunk.*" There are, of course, various gradations in the severity of the symptoms, in the lesser degrees of drunkenness. In some subjects, a sudden indulgence in considerable doses of alcoholic fluids—an outbreak into a debauch—excites a form of acute mental derangement—*mania a potu*, or acute alcoholic delirium*—which is confounded with *delirium tremens;* but for the production of the latter disease chronic changes due to alcohol are necessary. Acute alcoholic delirium, on the other hand, is due to the immediate impression of the alcohol on the brain of a susceptible subject—usually a young man having strong neurotic tendencies.

ACUTE ALCOHOLIC DELIRIUM, *or Mania a potu.*—This condition is usually confounded with *delirium tremens.* It differs from it, however, in that it is the direct result of alcoholic excess, in a subject free from the numerous changes of chronic alcoholism. Those suffering from this malady have been engaged in a sudden debauch, or have drunk liquors very deeply for a comparatively short time. Besides the sudden and great excess in drinking spirits, they have usually been subjected to some powerful mental excitement, to mental worry, to chagrin, etc. Under the influence of these causes, they grow more and more excited, become wakeful, lose their appetite, and presently become the prey of hallucinations. The delirium is similar in character to that of delirium tremens, but the trembling is wanting, the complications of the latter are not present, and the termination is earlier. The delirium may be as violent as that of delirium tremens, but it is not so important, and a prompt cure may be readily effected. It is true, now and then, that such a case terminates in mania when occurring in a subject having strong proclivities in that direction. Usually, the prompt withdrawal of the offending cause, proper alimentation, and cerebral sedatives, as the bromides and chloral, effect a speedy cure. In such cases, the question of the cessation of the spirits can not be for a moment doubtful. The effect being due to the impression of alcohol on the brain, no structural alterations having occurred, the obvious relief consists in the removal of the cause.

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* Magnan, "On Alcoholism," translated by Dr. Greenfield. London, H. K. Lewis, 1876.

CHRONIC ALCOHOLISM.—From the brief view of the changes wrought in chronic alcoholism, before given, it is sufficiently evident that these changes may be comprehended in two groups, sclerosis and steatosis. In the brain and nervous system, as elsewhere, disorders develop, indicating the greater or less progress in these morbid processes. In the intellectual, motor, and sensory sphere are they alike exhibited. With the progress of the affection, the memory grows weaker, the judgment becomes less accurate, and the power of attention and of association of ideas greatly diminishes. Hence the puerilities of thought, the rambling and incoherence which are characteristic of the alcoholic. His moral sense is blunted; his duties to his family and to his business are neglected; he grows indifferent to his personal appearance, and becomes dirty in his habits. To remove the feelings of discomfort, which come on when the influence of the spirit declines, a constantly increasing quantity is necessary. He becomes dejected, morose, and irritable, and more and more stimulant is required to lift him up from his wretched moral state. The appetite declines, and is confined to a taste for condiments, for stimulating articles, and for those having a strong, even a biting flavor. The stomach becomes intolerant of food, and vomiting frequently occurs. Especially does the alcoholic suffer in the early morning before the morning dram gives steadiness to his nerves and tone to his stomach. There is, then, much straining and retching, only some glairy mucus and a little greenish matter coming up after great anguish. The mind becomes more and more impaired, the conversation is a maudlin rambling, and ultimately the mental condition declines into imbecility.

As regards the exterior of the body, chronic alcoholics exist in two types: the pallid, flabby, but fat; the red, even purplish-hued, and bloated—the former having a smooth, pallid, heavy, and imbecile expression; the latter, roughened by pimples and stigmata, dusky, with great bladders under the eyes, yellow and injected conjunctivæ, and lips blue and swollen. Before these external features are well marked, the symptoms produced by the anatomical alterations occurring in all parts of the body are developing. The chronic alcoholics experience disturbances in the functions of various organs. They have more or less headache, or a sense of weight and oppression in the head, ringing and drumming sounds in the ears, and attacks of dizziness or actual vertigo. Vision grows dull, objects float before the eyes, they see flashes of light, and especially when about to fall asleep. Tremor now begins to be manifest, first probably in the lower extremities (Anstie), but soon occurring in both ; at first under control, so that a strong effort can quiet the muscles, but presently becoming uncontrollable. The trembling is conspicuously worse in the morning before the drink and food have had time to support the waning power. Numbness, tingling, paresis of the muscles, occurring in one member, or on one side, and of brief duration, are not uncommon. Sudden attacks of vertigo, with instantaneous loss of voluntary control, the patient falling, with or without loss of consciousness, are sometimes experienced. With such attacks there may be twitchings of the muscles of the face or of a member, when, of course, the seizures wear an epileptic aspect. Hallucinations are experienced at this period at the moment of falling asleep or on awaking. When the alcoholic subject has attained to this degree of development of his disorder as manifest in these nervous symptoms, and in the state of his bodily nutrition as already described, he presents characteristic symptoms of disorders of digestion. The tongue may be heavily coated, or glazed and fissured. The breath is fetid from the presence of products of alcoholic decomposition. The appetite for ordinary food is almost lost, and much distress is experienced after eating, but especially in the early morning. Vomiting of blood is not infrequent. The stools are much altered in character, are often fetid, black and tar-like in consistence, and not seldom consist of blood. Hæmorrhoids form and often bleed freely, and sometimes *fistula in ano* occurs. In consequence of the obstructive changes in the liver, ultimately effusion takes place in the abdomen (ascites), and œdema in the lower extremities. When hæmorrhages occur from the intestinal mucous membrane, ascites is not so likely to develop. Sclerotic and steatose changes occur in the heart ; the cavities are apt to dilate ; calcareous deposits take place in the valves ; the great vessels and the intra-cranial vessels undergo atheromatous degeneration, and cerebral hæmorrhage is one of the results which may be expected under these circumstances. Sclerosis and fatty changes may also occur in the kidney, and albuminuria result.

Chronic alcoholism tends to develop several distinct morbid states : an acute exacerbation called delirium tremens ; acute alcoholic mania ; acute alcoholic melancholia ; dipsomania ; acute alcoholic pneumonia. These require separate consideration, and with the fullness demanded by their relative importance.

DELIRIUM TREMENS.—Causes.—In the greatest number, delirium tremens is due to the action of the alcoholic fluid ; it is an acute alcoholic delirium due to an unusual consumption of spirits by the subject of chronic alcoholism. In a smaller number, it is caused by the sudden withdrawal of the accustomed stimulus ; the stomach is disturbed, food and drink are rejected, and hence the nervous system is left unsupplied. An attack may also be induced by some strong moral emotion or excitement, or by an accident or injury.

Pathological Anatomy.—The anatomical alterations are those of chronic alcoholism. The brain has the appearance characterized by morbid anatomists as the "wet brain"—that is, there is much fluid in the subarachnoid space, in the ventricles, and at the base, and the veins and sinus are distended, the substance of the brain itself being more or less œdematous. In some instances there is active hyperæmia, the red points are more numerous, and vessels not seen in the normal condition become prominent. Meningitis, cerebral hæmorrhage, etc., may be present as complications. The most important complication is, however, pneumonia. The condition of hypostasis should not be confounded with hepatization. Renal changes are by no means infrequent.

Symptoms.—A continuous debauch may inaugurate the symptoms, or the stomach become very irritable, the appetite is lost and even the drink is rejected. The trembling characterizing the ordinary state increases; the manner grows excited and irritable, and the countenance, before dull and apathetic, now appears animated and restless. Insomnia is an early symptom; but snatches of sleep are obtained, or the night is passed in the vain effort to get a moment's repose. Then, the characteristic hallucinations and illusions come on. A patient of the author's, while apparently well, began to suffer from wakefulness, and, coming to him in the hurried and excited way characteristic of this state, said, with an air of mystery but of entire conviction, "It's most extraordinary," taking off his hat, "but the story of the garden of Eden is all dramatized on my hat," and he proceeded to point out with much eagerness each detail, until I startled him by declaring it an hallucination. Very often, for several days, such a patient will be about, under the influence of some illusion in regard to his own occupation, or to some public or private affairs, or of some extravagant delusion. Sometimes his notions are gay and pleasing, and he is all hilarity, but more frequently they are gloomy and frightful. The beginning of the delirium is usually at the moment of falling asleep, or in awaking, when the insomnia first occurs. He then sees frightful objects—goblins, demons, and monsters—but, fully awake, they vanish, and he is able to appreciate his real position. This preliminary state is often called "the horrors." With the progress of the case the hallucinations become constant. The condition is that of fright; the patient is menaced by persons, or demons, who take his life and he seeks to escape. As any one may assume this shape, such a patient may be dangerous, for, although the delirium is cowardly and he seeks to escape, he may, on a sudden, if he have a weapon, do some mischief, or he may cast himself from a window. He sees objects on the wall, the figures of the paper appearing as snakes or devils, and they threaten and mock him. The figures on the coverlet appear as insects and snakes, and he tries to toss them off, or escape them. He peers furtively in the corners, and, rising up suddenly, looks under the bed. His eye rapidly glances about the apartment, and has a troubled and suspicious expression. He may be noisy and furious, yelling and screaming incoherently, fighting all who approach, and spitting out his food and drink. The quiet, cowardly and shrinking patients are usually controlled by firmness on the part of physician and attendants, but the more furious and maniacal may require the camisole. Besides the visual, there may be illusions of smell, the patient perceiving disgusting odors, and he may go about the apartment snuffling. Another has illusions of hearing, strange voices mocking or upbraiding or threatening him. In fact, the forms which his morbid fancies take are almost endless. The morbid activity increases the rate of circulation and excites sweating; but more or less fever comes on after a preliminary stage of depression. This stage of depression is characterized by a cold, clammy skin, a feeble pulse, and general muscular weakness. Fever then slowly develops; the temperature rises in some cases to 105° Fahr. (Magnan); the pulse becomes rapid, and is marked by an extreme dicrotism. The tongue is moist and tremulous, sometimes coated heavily, more frequently is merely pasty. The stomach continues irritable, and food, if swallowed, is rejected; but usually difficulty is experienced in feeding these patients, and, when delusions of poisoning exist, feeding can be accomplished only by mechanical means. The bowels are apt to be confined. The stools are often dark and offensive, sometimes blackish and tar-like. The urine is scanty, very high colored, and may contain albumen.

Course, Duration, and Termination.—The course of delirium tremens is usually acute. Complications may arise to terminate the case in a few days, as a double pneumonia, a cerebral hæmorrhage, etc.; but the ordinary duration rarely exceeds two weeks, by which time recovery or death will have taken place. The first stage, as it may be called, from the beginning of wakefulness and hallucinations to the rise of fever, is very variable in duration, and may last for a week or ten days. Convalescence is inaugurated when sleep occurs and the patient awakes refreshed, and, taking food, retains it, and at the same time becomes clear in mind. Short snatches of sleep, the delusions continuing, and food still rejected, do not mark the beginning of convalescence. If the delirium subsides, but the patient still mutters and picks at the bedclothes, the tongue becoming dry and cracked, and regurgitation of dark, brownish and bilious matters taking place, the condition is a bad one, and an early fatal termination may be expected. Sometimes death occurs suddenly from failure of the heart; in the midst of active delirium the pulse becomes rapid and thready, the surface cold and clammy, the features anxious and pinched, and death ensues in a few hours, or a few minutes even. Sometimes, after waking up from a state of forced sleep by narcotics, the patient passes into a condition of profound prostration which soon proves fatal.

Diagnosis.—The symptoms are so characteristic and the history so unequivocal, that an error is not likely to occur. Delirium tremens may, however, be confounded with its congeners, *acute mania* and *acute melancholia*, due to chronic alcoholism. The distinction rests on the characteristic trembling, the delirium of fear, and the peculiar hallucinations of delirium tremens, as well as its acuteness. The delirium which accompanies alcoholic pneumonia is like delirium tremens, but it arises during the pneumonia, whereas, when pneumonia complicates delirium tremens, it arises during the course of the latter.

Treatment.—There are two points to which attention must be directed : to provide suitable aliment ; to procure sound sleep. As the stomach is very irritable, milk and lime-water may be given freely but at regular intervals. If the attack has occurred in consequence of the failure to retain the spirit, it is advisable to give a moderate amount of whisky or brandy with the lime-water and milk. In old drunkards it is not unfrequently the case that no aliment will be appropriated unless some spirit is given with it. When this condition exists it is indispensable to allow a moderate quantity of whisky or brandy. Sometimes an egg will be eaten, beaten up in beer or ale, but more frequently than any similar compound aliment will egg-nogg or egg-flip be readily taken and assimilated. Beef-juice may be given in alternation with milk, and, if the stimulant is necessary, can be added to it. When the attack of delirium tremens has succeeded to an unusual consumption of liquors, they should be discontinued, or given in much less amount. Here, also, may exist the same state of the digestive function, and the same impossibility of procuring assimilation without the accustomed stimulant. In fact, in this circumstance lies the solution of the problem. Can digestion and assimilation proceed without the stimulant ? If so, it is unnecessary—for nothing has been more conclusively established than that the patient does well if he can take and appropriate sufficient aliment. The beef-juice or other animal broths given should be well fortified by red pepper, which serves a double purpose—to stimulate digestion and to act as a cerebral sedative. A bolus of capsicum, containing ʒ ss to ʒ j, every four hours, is now known to possess distinct sedative and hypnotic properties, and has been successfully used in the treatment of the disease. The notion, formerly entertained, that to procure sleep by large doses of opium is the only objective point in the treatment of delirium tremens, has happily been abandoned, for under this system many patients were either fatally narcotized, or forced into a condition of *coma vigil* terminating in collapse. Forcing sleep is secondary to careful alimentation. The best agent for securing sleep is chloral, or a combination of chloral and morphine; but chloral is not proper when the heart is weak, and opium or morphine when the tongue grows dry, and the delirium increases under its use. If, however, fifteen grains of chloral and one fourth of a grain of morphine secure sound and refreshing sleep for several hours, the patient awaking free from delusions, they have unquestionably done good. In the preliminary stage of "the horrors" sleep may be procured by full doses of bromide of potassium. Cardiac paralysis has ensued in several cases of delirium tremens, after the administration of chloral and bromide of potassium, and fatal narcosis by the combination of chloral and morphine. Pilocarpine has proved to be exceedingly effective in causing sleep and quieting delirium. When there is a decided tendency to cardiac failure, and at the same time active or furious delirium, tincture of digitalis in drachm-doses, or more, is unquestionably very beneficial. Where opium is not well borne, or contraindications to it are present, tincture of cannabis indica may be used with advantage. The internal use of chloroform has acted well in some cases in procuring sleep; but the inhalation of chloroform is very hazardous, and has proved fatal. Besides the dietetic and medicinal treatment, certain moral considerations must have due weight. The subject of delirium tremens should be in charge of a resolute and patient nurse. The apartment should be as remote as possible from the noises of the outside world. The walls should be of a neutral tint, without figures, and the bed-hangings, curtains, etc., should be perfectly plain and of some subdued color. All objects in the room not necessary to the care of the patient should be removed; as little as possible should his attention be attracted by coming and going, and all appearance of mystery, such as whispering, the exchange of signals, etc., should be avoided.

Acute Alcoholic Mania is an outbreak of acute mania due to alcoholic excess, and to the changes induced by such excess in the condition of the intra-cranial organs. The predisposition is inherited. The special point in such cases is the tendency to the commission of homicidal acts.

Acute Alcoholic Melancholia, like acute mania from the same cause, is induced by drink in a subject having an inherited tendency. The symptoms present the usual type, and the special characteristic is the desire of self-destruction.

Dipsomania, as the name imports, is that mental condition which impels to the drinking of intoxicating liquors. This form of mental disorder is the sad inheritance from drunken parents. At the earliest period after taste has become differentiated, these unfortunates display a strong and special inclination for liquor, and for its exhilarating effect, and by the time puberty is reached they are already drunkards. In some cases this mental disease manifests itself in periodical attacks, characterized by a ferocious and utterly uncontrollable impulse to indulge to excess in strong drink. These periodical attacks are at first separated by considerable intervals, and, beginning at puberty, may not seriously impair the tone of the mind and the power of self-control until thirty-five, but from this period on the intervals become very narrow, and the entire surrender to alcoholic excess follows at no distant time.

Acute Alcoholic Pneumonia.—The most frequent and fatal complication of delirium tremens is pneumonia; but the latter is very frequently mistaken for the former. In old alcoholics, an attack of croupous pneumonia approaches insidiously, and the first symptom indicating illness may be the peculiar hallucinations and illusions. Very often the hallucinations refer to the difficulty of breathing, the patient maintaining that the air is stuffed with something, or that something interferes with its entrance to his chest. The delirium under these circumstances is comparable to that which comes on in the inebriate after an injury or a surgical operation. The pneumonia not being recognized, the case appears to be one of delirium tremens. The radical distinction between the two affections is this: In acute alcoholic pneumonia, the pulmonary disease precedes the delirium tremens and is the cause of it; in delirium tremens, pneumonia is a frequent complication. In the treatment of acute alcoholic pneumonia, the habit of the system should not be broken off, but stimulants should be allowed, and they may be pushed freely.

Sequelæ of Chronic Alcoholism.—Besides the morbid states which may develop during the course of chronic alcoholism, there are sequelæ which require some consideration. We owe particularly to Magnan * the development of our knowledge on this point. It is not difficult to comprehend the relation of the various anatomical alterations produced by alcohol, and such consecutive maladies as ascites, dementia, general paralysis, and the mental disturbance produced by hæmatoma of the dura mater. Ascites, dementia paralytica, and hæmatoma, have been studied elsewhere, and the mental enfeeblement produced by atheroma of the cerebral vessels has been mentioned in connection with that topic. It is merely necessary here to name these sequelæ, and to invite the attention of the reader to their independent treatment under their appropriate heads.

Treatment of Chronic Alcoholism.—For the disorders of digestion, morning vomiting, and loss of appetite, accompanied by wakefulness and nervousness, the appropriate remedies are abstinence, careful alimentation, the administration of such tonics as quinine, tincture of nux vomica, oxide of zinc, etc., and the use of bromide of potassium to procure quiet sleep. In the more chronic cases, where degenerative changes may be expected to have taken place, arsenic in small doses

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* "On Alcoholism," etc., London, H. K. Lewis & Co., 1876.

(two drops of Fowler's solution *ter in die*), the compound sirup of the hypophosphites or sirup of the lacto-phosphate of lime, and cod-liver oil, are to be strongly commended. The phosphates and cod-liver oil should be taken for many months at a time. The chloride of gold and sodium and the corrosive chloride of mercury, the author believes, have the power to retard the changes in the connective tissue taking place in chronic alcoholismus. To effect any obvious results, they must be given before the changes are too far advanced, and must be continued in small quantity for a long period. With these measures must be conjoined a suitable hygiene, proper occupation, and abstinence from alcoholic beverages of all kinds.

AMYLOID DISEASE.

Definition.—*Amyloid disease* is a general condition in which a peculiar albuminous material is deposited from the vessels into the adjacent tissues. The deposition of this morbid material is usually preceded by chronic suppuration in some form. Various names have been applied to this disease, as colloid degeneration, lardaceous disease, waxy degeneration, bacon-like (*speckig*, of the Germans) degeneration, etc.

Causes.—Suppuration, in connection with caries of the bones, long continued, is the most frequent cause. By no means so often does suppuration of the soft parts produce the same result. Among the conditions of this kind are ulcers of the leg of long standing in elderly subjects, old fistulæ, rectal, urethral, and vesical, but especially empyema with or without fistulous communication externally, or by a bronchus. Bronchiectasis, phthisical cavities long suppurating, chronic abscesses, pyelitis and pyelonephritis, chronic dysentery, etc., have been followed, after a more or less protracted course, with amyloid disease. The vast extent of suppurating surface in phthisis, when the tubercular ulcerations occur in the larynx, bronchi, lungs, gastro-intestinal and genito-urinary mucous membrane, furnishes the occasion for amyloid disease in a considerable proportion of all the cases—in a majority of the cases, according to some observers (Schueppel). It is certain that amyloid degeneration is caused by the syphilitic cachexia, especially when there are ulcerations and prolonged suppuration in parts of the body, more certainly when the bones are affected. Hereditary syphilis is also sometimes a cause. There is much reason to believe that a protracted cachexia, produced by chronic malarial poisoning, has given rise to amyloid deposits. Although it appears necessary that suppuration be protracted, Cohnheim * has shown that, as regards gunshot wounds of bones, three months of suppuration, only, is sufficient. He records three cases in which death ensued after wounds of bone, in six, five, and three months respectively, the amyloid deposits being widely distributed.

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* Virchow's "Archiv," Band liv, p. 271; "Zur Kentniss der Amyloidentartung."

As regards sex, men are much more liable to the disease than women—the proportion being from two thirds to three fourths. It occurs at all periods in life, but is more frequent at the most active and exposed age.

Pathological Anatomy.—The term *amyloid*, or starch-like, is rather misleading, since the material is albuminoid in its characteristics. The *corpora amylacea*, as found in pathological products of the nervous system, present a strong resemblance to starch-granules, but the amyloid matter, as deposited in the cachexia, with which we are now concerned, is very different in appearance, and "probably has nothing in common" with those bodies (Wagner).*

It has not been possible, hitherto, to separate the amyloid deposit from the tissues in which it is deposited ; hence the published analyses are misleading. It is closely related to glycogen. It has the distinctive property that it resists the action of digestive ferments, and but slowly yields to putrefactive fermentation. Its presence in tissues is readily determined by the reagents originally proposed by Virchow —iodine and sulphuric acid. The suspected material is washed over with a solution of iodine in iodide of potassium, when the amyloid parts are stained of a mahogany brown, the healthy tissue appearing a faint yellowish tint. If dilute sulphuric acid (two *per cent.*, Kyber) is afterward brushed over, the amyloid matter assumes slowly a dark-blue color. The iodide of methylanilin has been brought forward lately as a test. Parts of the suspected organ (the liver, for example), hardened in alcohol, are placed in a solution of the iodide of methylanilin, and in a few minutes the amyloid deposits are stained a ruby-red, while the rest of the tissue presents a cloudy, bluish tint. This is less certain than iodine and sulphuric acid, is the judgment of Kyber.

In some way, not now understood, the albuminous body, which must exist in the blood prepared for tissue-formation, is precipitated in an insoluble form in the walls of the nutrient vessels or in the tissues adjacent. There are two theories now held in regard to the deposits : according to one, the deposits occur in the tissues adjacent to the vessels ; according to the other, into the walls of the vessels. Just about the vessels, and in their walls, is deposited a thick, colorless, waxy material which, as it solidifies, has the transparency, almost, of glass. The German name given to it signifies that this material resembles lard in appearance. The deposits do not follow any regular plan, and are variously distributed in the affected organs. The initial deposit takes place in the liver, and in that zone of the hepatic structure which is concerned, as is supposed, in the formation of glycogen. The branches of the hepatic artery are first attacked, then the capillaries of the lobules are invaded. According to Virchow and his followers, the deposits occur in the liver-cells, while Wagner maintains that the capillaries only are invaded, the liver-cells perishing by atrophy and fatty degeneration. Schueppel is inclined to agree with Wagner, but Kyber * affirms that the liver-cells are invaded by deposits, as he has isolated them and recognized the changes. As a result of the deposits the organ enlarges, sometimes enormously, reaching the weight in some instances of twelve pounds, and averaging twice the weight of the normal organ. The shape is not materially altered, except that the anterior edge is smooth, rounded, and thickened. In color, the organ assumes a light-grayish brown, or yellowish gray, and in consistence is very firm, resilient, or elastic, giving the impression of soft rubber, but not offering much resistance to the knife in section, although much tougher than the normal liver-tissue. The cut surface seems dry, is nearly bloodless, and has a grayish-yellow or brown color, like smoked bacon. The surface is not homogeneous, for the outlines of the acini are distinct, and the loblules are separated by a yellowish line. When the deposits are slight in extent, the whole organ will not appear changed, but in the middle zone of each lobule the amyloid infiltration first takes place, and here will be exhibited the peculiar grayish color and glassy translucency characteristic of the deposit. According, then, to the extent of the infiltration will the organ deviate from the normal. Instead of occupying a considerable portion of the liver, deposits may occur in spots forming nodular masses, the rest of the organ being healthy. Amyloid deposits in the liver may be associated with fatty degeneration of parts of the organ, with syphilitic nodules, and with metastatic abscesses.

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* "A Manual of General Pathology," by Prof. Dr. E. Wagner, *op. cit.*, p. 325.

In the spleen amyloid deposits are seen in two forms: in isolated, small nodules, formed about the Malpighian vessels—the *sago spleen*—and a general degeneration; the latter being merely a continuation and final development of the former. In the true amyloid spleen the whole organ is enlarged, firm in its structure, and having a light-brown or even grayish-yellow color. The cut surface is smooth and firm, and is not broken up into a pulp by pressure, as in the healthy organ. The trabeculæ are thickened and infiltrated by the amyloid material, giving to them the characteristic grayish appearance and vitreous luster. The deposits form thick and rather lustrous rings around the venous sinuses, and the walls of the larger and smaller arteries are infiltrated.

The amyloid kidney, like the amyloid liver and spleen, is larger and heavier than normal. Externally, the cortex is pale, even white, from anæmia, and has the peculiar glistening appearance characteristic of this degeneration. On section the same appearances are manifest, and the broad, whitish, translucent-looking cortex contrasts strongly with the dark cones. The vessels of the glomeruli are first changed; then the afferent arteries, and afterward the efferent, become the seat of considerable deposits which, however, are most extensive in the vasa recta. Subsequently the tubules and their epithelium are infiltrated. The supra-renal bodies, in a large proportion of cases—fifteen out of eighteen cases, according to Kyber—are affected in the course of the general disease. In extreme cases they are very large, pale, and waxy in consistence. The genito-urinary organs, the vessels of the mucous membrane of the ureters and bladder, of the uterus and prostate, and the muscular fibers of these organs, are also affected by amyloid change. The vessels of the mucous membrane of the tongue, œsophagus, stomach, and intestines, and sometimes the mucous membrane itself, are attacked. The arteries of the sub-mucous tissue of the intestines and the muscular layer of the stomach and intestines are also affected.

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* "Weitere Untersuchungen über die amyloide Degeneration," Virchow's "Archiv," vol. lxxxi, p. 1, *et seq.*

In a considerable proportion of cases the endocardium and the great vessels are affected, as well as the vessels of the body generally. The inner coat of the large arteries and the middle coat of the smaller arteries and veins are the parts invaded by the deposits (Kyber). We possess no information in regard to amyloid degeneration of the nervous system.

Symptoms.—When the various organs are affected by the lardaceous disease, a peculiar constitutional state—a cachexia—is induced. Previous to the deposition of amyloid matter, the vital forces are depressed by chronic suppuration, by the syphilitic cachexia, by chronic malarial poisoning, etc. Persons attacked with this disease are already pale, sallow, thin, weak, and depressed, in varying degrees, according as exhausting suppuration or syphilitic lesions or malarial toxæmia precedes its development. When the intestinal canal becomes affected, the appetite declines, there is considerable nausea, sometimes vomiting. As changes in the liver occur, hyperæmia and a catarrhal state of the mucous membrane are constantly maintained. Under these circumstances thrombosis of a stomach-vein may occur, resulting in ulceration, usually about the pylorus, and then vomiting of blood takes place. An intractable diarrhœa, colliquative in character, also comes on. The discharges are thin, offensive, very light or very dark in color; the latter, when blood is present. Particles of food, undigested and decomposing, are present in the evacuations, and ultimately such food as beef-tea, even milk, pass largely unchanged. Sometimes the stools appear like rice-water, are mucilaginous, and have little odor.

The clinical feature of amyloid liver is a persistent enlargement of the organ. As the weight of the liver, in extreme cases, may reach twelve pounds, it is usual to ascertain that the organ extends beyond its ordinary boundaries, to the breadth of the fingers, or more, below the inferior margin of the ribs. When thus enlarged, it can be readily felt through the abdominal parietes as of almost stony hardness. Sometimes the inferior edge of the liver may be grasped, and its condition noted, through the relaxed walls. Besides its hardness, the liver feels smooth, and is free from tenderness. It is but rare that jaundice occurs. Ascites is usual, and there may be general œdema or dropsy from the accompanying renal lesions. When ascites is alone present, there may be œdema of the feet and legs. Often the dropsy assumes a high grade; the abdomen becomes enormously distended, the scrotum attains vast dimensions, and the whole body is flooded with its own fluids.

Another large and hard body—the spleen—can also be made out in the abdomen. As the spleen is damaged, the anæmia increases. When the deposits are most pronounced in this organ, and when the disease begins its course by the changes in the spleen, an increasing leucocythemia is the most prominent symptom. When, however, the splenic disease is only a part of the general changes, and comes on after the lesions are well advanced elsewhere, there will be little alteration in the course of the malady except the more pronounced anæmia; but in the absence of ascites the splenic tumor may be made out.

The amyloid degeneration of the kidneys is manifest in the changes of the urinary secretion. As a rule, the amount of urine passed is great, except when a profuse, watery diarrhœa carries off the surplus fluid. The urine is, also, pale and of low specific gravity, when abundant, appearing to be little more than water. When scanty, the urine becomes dark and the specific gravity high. It may thus fluctuate between the extremes 1002 and 1035. The urea, chlorides, and phosphates are in very small quantity when the amount of urine voided is large, and relatively considerable when the urine passed is small in quantity. As the disease progresses, the relative quantity of solid matter in the urine diminishes. Albumen early appears in the urine, at first occasionally, as a mere trace, but, as the deposits increase, in constantly enlarging quantity, although never rising to the percentage of certain acute affections. Besides serum-albumen, the urine of amyloid kidneys contains a considerable quantity of globulin. Casts are only occasionally found, and are narrow and of the pale, hyaline variety. When the urine is condensed, dark in color, and loaded with albumen, which is an exceptional condition, the casts will be more abundant, chiefly of the narrow, hyaline kind, but also of highly refracting, yellowish, waxy material. General dropsy is comparatively infrequent, while ascites and œdema of the inferior extremities are common. Symptoms of uræmic intoxication are only rarely present, and for the obvious reason that the elimination of the urinary constituents, including urea, is not materially lessened by the amyloid degeneration. Even at the end when the quantity of urine passed is extremely scanty, there are but rarely slight symptoms of cerebral derangement, including headache, drowsiness, failure of memory, muscular weakness and tremors, and twitching of the facial and other muscular groups.

Diagnosis.—Amyloid disease, coming on in the course of some chronic malady with suppuration, can usually be readily diagnosticated. The coincident enlargement of the liver and spleen, the occurrence of polyuria with traces of albumen, and the troublesome diarrhœa, make up a morbid complexus of a very significant character. The enlarged liver may be confounded with the first stage of sclerosis or with echinococcus cysts. As regards the first, it is to be noted that the history is different, but the chief distinction lies in the fact that in sclerosis the enlargement is slight and is transient, whereas in amyloid disease it is considerable and persistent. In sclerosis the liver is irregular in outline and somewhat tender; in amyloid disease it is hard and without tenderness. The renal affection of amyloid disease may be confounded with interstitial nephritis. The history of the case becomes very important as a means of differentiation. In sclerosis of the kidney the albumen is more abundant; a marked reduction in the amount of urine, after a considerable increase, is observed; and cerebral symptoms are much more constant. The character of the casts, and especially the appearance of the yellow, highly refracting casts, responding to the iodine reaction, are of great value in the differentiation.

Treatment.—There are obvious indications for treatment in the debility and wasting with which the disease begins and is attended throughout its course. A full diet, rich in all the materials of nutrition, and especially of fats, should be prescribed. As the intestinal canal may be much damaged, foods converted into peptones in the stomach, and the aids to stomach digestion, acids and pepsin, are necessary. For the diarrhœa, two or three drops of Fowler's solution and twice the quantity of tincture of opium, persistently used, are probably the most efficient remedy. Hope's mixture, or an extemporaneous combination of nitro-muriatic acid and tincture of opium in camphor-water, may be substituted. Bismuth in full doses, with aromatic powder, is a useful astringent. Without a careful regulation of the diet, no remedies will succeed. Cod-liver oil, as a food and restorative, will sometimes improve the diarrhœa. As remedies for the local deposits —to convert the insoluble albuminous material into a soluble—phosphorus, the phosphites, and phosphates, are, in the author's experience, entitled to the first place as remedies. Phosphorus in minute quantity ($\frac{1}{120}$ grain), dissolved in cod-liver oil, is probably first. Phosphites, in the form of the compound sirup, is a valuable combination. The phosphates rank next. The author has had excellent results from the phosphate of soda in cases without diarrhœa, which is a contra-indication to its use. These remedies should be used persistently and for lengthened periods. The iodide of potassium, with a generous diet and iron, is a method of treatment strongly urged by Bartels, and with which he has, he says, succeeded in effecting a cure. A careful mercurial course, with or without conjoint or alternate administration of the iodide of potassium, may be useful in cases originating in the syphilitic cachexia. Dickinson, influenced by his theory of amyloid deposits, administers alkalies, which restore their solubility. Alkalies may be serviceable, but the depression caused by them should be prevented by the timely administration of iron and a generous diet.

ANIMAL POISONS.

HYDROPHOBIA.

Definition.—*Hydrophobia* is a specific disease due to the inoculation of a poison contained in the saliva of rabid animals, notably the dog, and characterized by pain and stiffness of the inoculated part; by exaltation of the reflex faculty; by spasms of the throat on the attempt to swallow, and subsequently at the sight of liquids; by delirium, exhaustion, and death. It is also known as *rabies canina.*

Causes.—The sole condition necessary for the causation of hydrophobia is the inoculation of man with a contagious principle contained in the saliva of the dog, cat, wolf, and some other rabid animals. This principle is not absorbed through the unbroken skin, but from a wound or abrasion. A certain predisposition is also necessary, it is probable, for, of all bitten by animals unquestionably rabid, but a small proportion are attacked by hydrophobia. The proportion is variously stated from five to fifty per cent., but, while the former is much too small, the latter is excessive. Accident more than predisposition is, however, the real cause of the exemption of so many who are bitten. The teeth, in inflicting the wound, pass through clothing, which removes the saliva, and hence the most of those bitten through the clothing escape infection. On the other hand, wounds of exposed parts, or an abrasion receiving the saliva, is very certain to be followed by the disease, unless there be a decided insusceptibility to the action of the poison. All ages and both sexes are liable, but more men than women are attacked, because the former are more exposed. Various moral impressions favor the occurrence of the disease. These are apprehension, fear, excesses of all kind, fatigue, etc.

Pathological Anatomy.—There are but few changes found *post mortem* really typical, if any such exist, but are common to all the diseases of the same group. The cadaveric rigidity is well marked; there are extensive suggillations, and putrefaction soon begins; the coloring matter of the blood stains the vessel-walls, and the blood itself is fluid and has a violaceous color. These facts only indicate a changed state of the blood common to many maladies. The fauces are red and swollen, the salivary glands enlarged; the trachea and bronchi are hyperæmic and contain a quantity of frothy mucus; the lungs are also hyperæmic and sometimes œdematous. More or less congestion of the brain, effusion into the ventricles, and hyperæmia, with enlargement of the vessels of the medulla oblongata, have been observed. In some cases changes of texture, softening, etc., have been seen at the apparent origins of the seventh, eighth, and ninth nerves. The pneumogastric, phrenic, and sympathetic nerves have also been found in a more or less hyperæmic state.

Symptoms.—The period of incubation is by no means confined to fixed limits. In 214 cases collected by Jaccoud, the period of incubation was less than one month in one fourth of the number, from one to three months in 143, from three to six months in 30, and from six months to a year in 11. According to Gamgee, in the large majority of cases, the period of incubation is four to eight weeks. Age apparently affects the duration of this period. Thus in nine new-born infants, the incubation period was thirteen to fifteen days. A very remarkable case has been reported of a man two years in prison, who had hydrophobia, and who had been bitten seven years before. During the period of incubation there is nothing in the wound, nothing in the state of the organism, to indicate the existence of any mischief. The wound or abrasion may be very slight, may have healed long since and been forgotten. At the termination of the incubation, the attention of the patient is attracted to the wound by some uneasiness felt in it. If it has not healed, the wound takes on a livid appearance, and becomes exceedingly painful, the pain shooting toward the trunk from the extremities if the wound is so situated. If the wound has cicatrized, the scar becomes painful, red, irritable, swollen, and sometimes exudes a bloody serosity. Sometimes a sensation of coldness and of numbness is felt in the bitten member, and occasionally the lymphatics of the limb are swollen, and marked by hard, red lines. The local symptoms are soon accompanied by systemic disturbances. The patient is depressed, apprehensive, peevish. So marked is the condition of melancholy that the first stage of hydrophobia has been called the *stadium melancholicum.* The skin becomes hot, the pulse rapid and bounding.

The appetite goes, and the bowels are confined. In some few cases the wound continues unaffected, and the feelings of anxiety and alarm are absent, the only symptoms coming on being the fever and the general distress belonging to the feverish state. What form soever this initial stage assumes, it is of short duration, continuing but a few hours or a day or two. The peculiar reflex paroxysms then come on: the breathing is sighing and jerking, the epigastrium is elevated by the forced depression of the diaphragm, and the shoulders are rendered prominent by the overaction of the levator and trapezius, while at the same time there is experienced a sensation of præcordial oppression and of tension in the anterior wall of the thorax. The neck grows stiff, the throat feels constricted, and the movements of the head are constrained. Now are experienced the peculiar sensations which are so distinctive of the disease. A spasm seizes the pharyngeal muscles when any attempt is made to swallow. The patient has an intense thirst, but whenever he approaches the cup to his lips his countenance assumes a strange expression, the eyes stand prominent, the features contract, the limbs tremble, and especially his hand carrying the cup, and he tries with a sudden movement to gulp down the liquid, but he can not pass it into the pharynx; it is violently rejected with a suffocative spasm, and he falls back on the bed exhausted. Presently, the appearance of water, the reflection from a mirror, any impression suggesting the act of swallowing, throws him into a state of apprehension or excites pharyngeal spasm. Meanwhile a sense of constriction continues at the throat, the mouth is dry and parched, and he is continually impelled to eject from his fauces, with a harsh, barking hawk, some viscid saliva. It is this hawking which is vulgarly supposed to be the bark of a dog. It must be admitted that this is a peculiar, unearthly hawking, which, under the circumstances, might seem like the bark of a dog. The appearance of the patient at this time is most striking. He is restless, his countenance anxious, his eyes bright and wandering; he becomes garrulous, and his mind presently wanders, and every few minutes he hawks and pulls at his throat as if to remove some obstruction. He will not tolerate the suggestion of liquids, much less their approach, and assumes a hostile attitude if there is a persistent attempt to induce him to try to drink. On the other hand, the mind may be clear, but this must be regarded as exceptional, for, in all the cases seen by the author, the patients, if not maniacal, were at least disordered in mind. Cases have been reported, however, in which the faculties of the mind were preserved, in which the patients not only were fully aware of their desperate condition, but expressed the greatest solicitude for their families and for those about them. The author has heard of one case in which the patient voluntarily asked to be restrained during the paroxysm, that he might not do injury to his attendants. There ensues such an exalted condition of the reflex faculty, at last, that a breath of air will excite the paroxysms, which are not unlike those of tetanus. When they come on, respiration is jerking, and then fixed, the voluntary muscles are rigid, breathing is suspended, the surface becomes red and cyanosed, and the action of the heart is rapid and weak. They last but a few seconds at first, but increase in duration and severity, and are excited by less and less powerful impressions toward the end. Sometimes there are severe and persistent erections (priapism), and in women there is nymphomania. Difficult urination is not uncommon, and in some cases strangury is present.

Course, Duration, and Termination.—Hydrophobia is a very acute disease. The first stage does not exceed two or three days, and may be but a few hours in duration, the average being about one day. The duration of the second or hydrophobic stage is similar; it may last two days, possibly three, but it is usually ended in one, sometimes in a few hours. The termination may be by exhaustion, the under jaw drops and the saliva flows from the corner of the mouth; the pulse becomes small, weak, and thready, the body is covered with a cold sweat, the pupils are dilated, the eyes fixed, the voice fails, and the patient, after a short, convulsive trembling, passes into collapse, and dies. In other cases the patient dies asphyxiated in the paroxysm. In still others, general convulsions end the case. The whole duration of the disease is comprehended in about three days. The prognosis of hydrophobia is most unfavorable, no cases of the genuine disease having ever recovered, unless we may except two, treated with woorara, lately reported.

Diagnosis.—There is a strong resemblance between tetanus and hydrophobia: in both the reflex function of the spinal cord is highly excited, in both slight peripheric irritation excites spasms; but they differ in that hydrophobia follows a bite of a rabid animal after a long period of incubation, and tetanus is caused by a wound; in hydrophobia there is a sense of constriction of the fauces—in tetanus there is trismus; hydrophobia is of much shorter duration than tetanus, is invariably fatal, while a considerable proportion of the cases of tetanus get well. Hydrophobia may be confounded with an hysterical malady simulating it, but the latter is accompanied by other hysterical symptoms, does not prove fatal, and there is no history of the bite of a rabid animal. There are those who maintain that hydrophobia—as a disease due to a peculiar poison contained in the saliva of the rabid dog—has no real existence; that the poison is a fiction, and that the symptoms supposed to be produced by it are really due to the influence of sympathy, to the faculty of imitation, and to the imagination, the whole being intensified by morbid fears. It would seem impossible, on this hypothesis, to account for the occurrence of this disease in infants after being bitten. As, however, the imaginary disease is just as fatal as the supposed genuine affection, the practical physician will be indifferent to the theories, and will be as loath to encounter the one as the other.

Treatment.—When the bite of a rabid animal has been received, the wound should be scarified, cauterized with a hot iron, or every part of it touched with nitrate of silver. The success of Mr. Youatt has been so great with the nitrate of silver that severer applications would seem to be unnecessary. Permanganate of potassium, having succeeded so well in the bites of venomous snakes, should be tried. There is no specific to prevent the disease, and we are equally ignorant of a remedy to cure it, unless Pasteur's inoculations with the modified virus prove to have the effect which he claims for the method. Of all the remedies hitherto proposed, curare is the only one which seems to possess any power over hydrophobia. Two cases have been reported recently—one in Italy and one in New York—in which a disease, diagnosticated as hydrophobia by eminent practitioners, got well under the hypodermatic injections of curare. Chloral, chloroform, gelsemium, nicotine, etc., may be used to alleviate the distress.

PARASITES.

TRICHINÆ AND TRICHINOSIS.

Trichina.—This dangerous parasite is found in two forms, as the *intestinal trichina* which is sexually mature, and as the *muscle trichina*, not fully developed, or sexually immature. The name given by Professor Owen (*Trichina spiralis*) is based on the hair-like appearance of the parasite and the spiral form assumed by it in the muscular tissue. It is a very small, hair-like worm, having a head smaller than the rest of the body, while the caudal extremity is rounded. The females are three or four millimetres long, and contain a sexual apparatus consisting of an ovary, a uterus, and a vagina. Only a part of the sexual apparatus exists in the muscle-trichina, the rest being developed after the parasite has entered the intestinal canal of its host. It is viviparous, and discharges from the vaginal outlet about one hundred embryos a week, and the birth of the embryos begins in about a week after the female enters the intestine. As more females than males are born, and as successive formation of embryos from the eggs may take place,[*] the number developed becomes enormous. The male trichina is one half the size of the female, and contains at its caudal extremity the sexual apparatus. The viable embryos discharged from the female are in lively motion. They do not remain in the intestine, but begin a process of migration which only terminates when they have reached their habitat in the voluntary muscles. The manner of reaching their destination is not known—whether by the blood-vessels, by the lymph-channels, or by direct effort boring through the intervening tissues until the muscles are reached. As they have repeatedly been found in the blood and lymph,[†] and in the connective tissue only adjacent to muscles,[‡] and as the rate of migration is so rapid, it seems pretty certain that the distribution is chiefly passive by the blood and lymph-streams. Endowed with a strange instinct, these parasites, when they reach the muscular tissue, stop their wanderings, pierce the muscles, and force their way into the primitive fasciculi, where they coil up. The sarcolemma of the primitive fasciculus now undergoes thickening, a quantity of granular matter surrounds the parasite, and a number of "oval, vesicular-shaped muscle nuclei"[§] develop on the inner surface of the capsule formed by the thickened sarcolemma. In the process of transplantation of the parasite from the intestinal canal to the muscle, the parasite grows; but it reaches the greatest size in fourteen days after it is established in the muscle. In the intestinal canal the embryos have a very short lease of life (five to eight weeks); but, safely deposited in the muscle, they continue during the life of their host and for a short period after his death. In the muscles, after a time, the trichinæ undergo a further change. Lime-salts are deposited in and about the capsule, and ultimately in the parasite itself, when minute bits of lime, just visible to the eye, are seen more or less thickly distributed through the muscular tissue. The distribution of trichina is determined by the migrations of its hosts—the hog, the rat, and man. This parasite has been found in the cat and other animals, and has been artificially reared in rabbits and Guinea-pigs. In the dog, however, it appears to develop no further than intestinal trichina, migration of the embryos not taking place in this animal. As man and the other hosts of the parasite are to be found everywhere, so this parasite is universal. It is especially frequent in this country in the great West, because of the enormous extent of the pork-traffic. The proportion of hogs infected in the West is variously stated, but it is probably not an exaggeration to say that from one to twenty per cent. contain trichinæ.*

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[*] Cohnheim, "Zur pathologischen Anatomie der Trichinenkrankheit," Virchow's "Archiv," Band xxxvi, p. 163.

[†] Virchow, ibid., Band xxxii, s. 332, "Zur Trichinenlehre," contains also a full historical account of progress of discovery.

[‡] Ibid., Band xxxiv, s. 469.

[§] Heller, Ziemssen's "Cyclopædia," article "Migratory Parasites," vol. iii.

Trichinosis.—The symptoms produced by trichina, when these parasites reach the body of man, are entitled *trichinosis*. They are not very uniform, but a division into stages, based on the several steps in the life-history of the trichina, will be convenient. These stages are *the intestinal*, *the migration*, and *the encapsulation*. When a piece of pork, containing in every cubic inch eighty thousand (Dr. Sutton) trichinæ, is swallowed, these parasites are set free; they then complete their sexual development, and, as each female discharges a hundred embryos, the intestinal canal soon contains thousands. If few in number, there may be but little disturbance of the canal, but usually more or less irritation of the stomach and intestines follows in a short time after the infected meat is swallowed. In a few hours or in a day or two, some uneasiness of the stomach is felt, and in some cases severe attacks of neuralgia of the solar plexus; nausea comes on, and then vomiting occurs. The vomiting may end with the first effort which empties the stomach, or it may continue with much retching and anguish for several days. The mouth feels pasty, and a subjective sense of foul odor is also experienced. Intestinal uneasiness comes on with the irritability of the stomach; colic, more or less distention of the abdomen, and diarrhœa follow. The stools, at first composed of fæces merely, become watery, light in color, and may ultimately assume a nearly rice-water appearance. This symptom is more persistent than the vomiting, may continue, indeed, for several weeks, and is apt to be exhausting. Diarrhœa may alternate with constipation; in some cases there is constipation only. When the digestive disorders have persisted for several days, fever comes on in the usual way, preceded by shivering or a chill. It is probable that the fever is about coincident with the birth of the embryos and the beginning migration. The fever is remittent in type in the sense that typhoid fever is, which it closely resembles. In some cases the type is truly remittent, with a decided morning remission and an evening exacerbation. The pulse is quick, rather small, and early shows a tendency to weakness, the range being from 90 to 140. There is intense thirst, the tongue and lips are dry, and the face is red and swollen (Davaine). During the existence of these symptoms the muscles of the body generally are sore to the touch and are flabby, but this state must not be confounded with that condition of the muscles caused by the migration of the parasites into them (Heller). The *migration period* is especially marked by the invasion of the muscles. The symptoms due to this invasion do not occur earlier than the tenth day, which allows three days for the migration from the intestine. The muscles are affected to varying degrees of severity, doubtless, according to the number of parasites entering them. There may be only a little soreness, but in decided cases, as might be expected, the muscles are hard, swollen, and very tender. The muscles of the extremities, especially the flexors, are penetrated, but those of the trunk also, only to a less extent. In consequence of this, the muscles are the seat of violent rheumatoid pains, and motion increases the distress. Hence the patients lie motionless, with the limbs semiflexed. As the muscles of mastication and deglutition are also invaded, chewing and swallowing become difficult and painful; hearing is impaired because of invasion of the stapedius muscle, and vision may be double or distorted because of the penetration of the ocular muscles. Œdema of the eyelids is one of the first symptoms of this period, and subsequently œdema of the extremities and effusion into the peritoneal cavity appear. For the same reason, doubtless, that the voluntary movements are impaired, the respiration is embarrassed, and dyspnœa is added to the other difficulties, and by the end of the fourth week a general bronchitis, a pleurisy, or a pneumonia may arise to complicate the case.* During the development of these formidable symptoms, the mind may continue undisturbed; in fact, a singular apathy takes possession; in other cases delirium occurs, but this may result from the wakefulness, the *coma vigil*, which is so pronounced a feature of the cerebral condition in many adults. In children there is a condition of somnolence throughout. Various miliary and pustular eruptions appear on the skin, which is extremely sensitive, but the most pronounced symptom connected with this organ is the profuse sweats which appear early and continue throughout the disease. The sweats are not critical, and do not modify the temperature. Bed-sores form to a slight extent, and desquamation of the cuticle occurs during convalescence. Abortion sometimes takes place, but the fœtus is free from trichinæ; and, on the other hand, pregnancy may continue undisturbed. The menses may or may not be interfered with, more usually not. *The course* of trichinosis is greatly influenced by the number of parasites. A small number may cause a mere temporary diarrhœa; a large number may produce a violent gastro-enteritis, sufficient to cause death without the migration into the muscular system (cases by Dr. Sutton). In such cases there will occur the symptoms of gastro-enteritis only, and, after death, intense hyperæmia, swelling of the mucous membrane, and destruction of epithelium will be seen. The range of temperature in these cases is from 98° to 100°, and the type of the fever remittent. When migration of a small number of parasites occurs, the fever will assume the typhoid aspect, the temperature range from 100° to 104°, the usual muscular soreness to a small extent be felt, but the most pronounced symptoms will be those of inflammation of the gastro-intestinal canal. Recovery may ensue in such a case by the encapsulation of the parasites, and a gradual subsidence of the gastro-enteritis. From three to four months will be occupied with such a case from its beginning to the completion of convalescence. In the severest cases all the symptoms given above will appear, and death will take place in three to four weeks, frequently caused by pneumonia. The mortality from trichinosis will range from twenty to fifty per cent., dependent of course on the amount eaten of any given specimen of trichinous pork.

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* The reader is advised to consult an excellent paper by Dr. Sutton, of Aurora, Indiana, giving an account of an outbreak of trichinosis, and some general remarks on the proportion of trichinous pork, which he puts at three to sixteen per cent. for southeastern Indiana. (Reprinted from "Transactions of the Indiana State Medical Society.")

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* Davaine, *op. cit.*, p. 760.

Diagnosis.—Cases of trichinosis are often mistaken for ileo-colitis and for typhoid fever. From the former it may be differentiated by the œdema of the eyelids, the muscular pains, and the profuse sweats. The range of temperature being much the same as that of typhoid, the distinction between the two must rest on the muscular symptoms, the œdema, the pain and hyperæsthesia, the profuse sweats, and the absence of the muttering delirium, the subsultus, and other nervous symptoms. The œdema occurring in this disease, which is general, is separated from cardiac and renal dropsy by the absence of cardiac and renal disease, and by the other symptoms pertaining to trichinosis. In cases of doubt, the harpoon may be used to take out a bit of muscular tissue for examination, but this is a measure of doubtful propriety, because severe gastro-enteritis may ensue without migration. In typical cases the harpoon would hardly be necessary, yet Dr. Sutton, removing a small piece of the gastrocnemius in one of his fatal cases, found it swarming with trichinæ, "estimated at more than one hundred thousand to the square inch," and they were in active motion, "coiling and uncoiling."

Treatment.—Attention should be at once directed to the destruction and removal of trichinæ in the intestinal canal. A variety of remedies have been proposed, but no success seems to have attended any of them, unless glycerine may be excepted. The vomiting and purging, if not excessive, should be promoted by diluents. Glycerine and water, which has the power to cause shriveling and death of the parasite, may then be given—one part of glycerine to two parts of water. Carbolic acid may be administered both with the view to allay the intense irritation and to act on the embryos. We venture to suggest a trial of carbolic acid and tincture of iodine for the same purpose. Corrosive sublimate, arsenic, picric acid, benzine, and other agents have been used to destroy the parasites in the intestines, but without results (Haller). Quinine seemed to exercise a good influence in Sutton's cases, the best, indeed, of any of the agents used. As this remedy has a toxic influence on the low forms of life, it seems desirable to employ it more freely in future cases. If constipation be the condition, purgatives should be administered without delay. The treatment to be pursued when the parasites migrate, must be purely symptomatic. The obstinate wakefulness and the pains will require morphine and chloral. Quinine and stimulants will be needed to support the powers of life. Milk, beef-juice, egg-nogg, and other aliment must be carefully administered from the beginning. There is but one point in prophylaxis. Meat containing trichinæ should be thoroughly cooked. As the cases arising from these parasites are caused by the consumption of raw hams and raw sausage recently cured, this practice should be totally discontinued.

CHYLURIA.

Definition.—By the term *chyluria*, it is intended to express a condition of disease, characterized by the presence of chylous matter in the urine. The milky or chylous appearance of the urine is now known to be due to the presence, in the blood and urine, of a parasite —*Filaria sanguinis hominis*—or a filaria having the blood of man for its habitat. This filaria is a nematoid entozoön, and hence this malady is appropriately classified with the parasitic affections. It is often confounded with ordinary hæmaturia, or bloody urine.

Causes.—Chyluria is a disease of tropical and sub-tropical regions, but cases have been observed in England, and they are not infrequent in the southern parts of this country. The West Indies is a favorite locality for the appearance of this parasite; and Brazil—as might be expected—has furnished many cases of chylous urine.

The parasite, to the presence of which, in the blood and urine, the disease seems to be due, is a nematoid entozoön having the diameter of a red blood-globule (=0·007 mm.) and a length of $\frac{1}{75}''$ (=0·34 mm.) (Lewis). The filaria is inclosed in an extremely delicate and transparent membranous sheath, and is, when living, employed in movements, now coiled, now fully extended. The relation of the parasite to the production of chylous urine is not clear, but the constancy of the association is undoubted, and various theories have been put forward to explain the presence of the lymph-fluid in the blood. By Robin and Bernard it is maintained that the state of the urine is due to the admission into the blood of the chyle, by means of the agency of the parasite. Dr. Carter holds that a direct communication is established between the lymph-canals and blood-vessels, but he does not explain how the parasite effects this admixture. Dr. Roberts also proposes an explanation, by which the chyle gets into the blood through an opening made by ulceration into hypertrophied lymph-canals. None of these theories reconciles all the facts, hence they can not be regarded as adequate. The constant presence of the filaria seems now to be well established; but the exact relation of the parasite to the chylous

urine remains to be demonstrated. Besides the
hæmaturia, a fluke was discovered in 1851 by D
ing the same condition.

Pathological Anatomy.—No disease of the k
covered, and, indeed, no change in any organ or
out, that properly pertains to the condition of
however, in all parts of the vascular system—
venous and arterial—numerous specimens of the
is also contained in the lymph-fluid discharged
the chylous urine. It follows, therefore, that the
induced by the parasite are the sole pathological
ria. As this state may exist for many years, var
eases can arise, but then the morbid conditions a
chylous state of the urine.

Symptoms.—It is now well known that there i
symptomatic expression of chyluria. Neverthel
of the author to present as clear an outline, as
toms properly pertaining to this state.

There may be no symptoms to indicate the
until the characteristic chylous urine makes its a
majority of cases there occurs some uneasiness in
through the loins, along the course of the uret
næum. Coincidently with this symptom there a
depression of mind. Hypertrophied lymph-ve
lymph-fluid escape, in the axilla, groin, scrotum,
casionally elephantiasis lymphangiectodes is asso
or, indeed, true elephantiasis may occur simult
characteristic symptom, however, is the presenc
the urine. When this admixture takes place, the
whey-like appearance and odor, and, after stan
coagulates, forming a jelly-like mass. If blood
times happens, the coagulated mass has a reddish
shreds of coagula, is seen at the bottom of t
Sometimes the contained blood coagulates in
structing the urethra, suddenly stops the flow of

The presence of chyle in the urine may be dete
by the use of ether to dissolve out the fatty m
precipitate the albumen. When the urine is sh
the fat is dissolved out, and hence the milky appe
boiled and nitric acid added, the albumen prese
important to note that the quantity of chylous
urine is not always the same : indeed, under s
may be absent. In different examples of the san
same case at different times, the quantity of emuls
greatly. There may be as much as 1·39 of fat

time, and in the same subject, a few hours subsequently, none may be detected. The fat and albumen are not always present in the same relative proportion. After active exercise, the albumen exists in greater quantity, and, after meals, the fatty matter. The latter may be nearly entirely absent in the early morning before any food has been taken, and must vary in quantity with the proportion of fatty matter in the food. Any analysis of the urine must, therefore, be considered with reference to the period at which the examination was made. In general, it may be said that chylous urine does not correspond in composition to any of the fluids of the body, but compares more nearly to chyle than any other.

The composition of the blood is, also, altered in chyluria. The proportion of fatty matter in normal blood is as 1 to 116 of albuminoids, but in chyluria the fat exceeds the albumen. Although there are discrepant opinions, the general truth is in the direction stated. Besides the change in the quantity of fat, the blood is not altered, except in regard to the presence of the hæmatozoön—the *Filaria sanguinis hominis*. The same parasite is found in the urine also.

Course, Duration, and Termination.—The course of chyluria is essentially chronic, and no fixed duration can be assigned to any case. The disease lasts from one year to fifty years. Now and then a case terminates fatally, in a most unexpected manner. The sufferers from this malady appear debilitated, and experience a marked degree of mental depression. The explanation of these symptoms may be found in the presence of blood or chyle in the urine. In some cases, without any apparent reasons, the urine assumes a milky appearance, and afterward the general symptoms of depression come on. Indeed, the aspects of the malady are Protean, and a proper discrimination is possible only by recognizing the filaria in the blood and urine. Even before the symptoms appear, sometimes filariæ can be detected. The prognosis can not be regarded as favorable in any case, since, when a cure has been apparently effected, the disease unexpectedly recurs, and this may happen many times.

Treatment.—No curative remedy has thus far been proposed. Gallic acid, in full doses—a drachm or two daily—has proved useful in some cases. Probably carbolic acid and tincture of iodine, alone or in combination, will be found to act beneficially. Large doses of iodide of potassium have proved beneficial in a few cases. Full doses of quinine may have an effect on the growth and development of the parasite. The influence of change of associations, of occupation, of climate, has not been sufficient to warrant the recommendation of such hygienic means. As the chronicity of the cases will permit, the various parasiticides may be tried in turn.

DISEASE-PRODUCING MICROSCOPIC ORGANISMS.

Forms and Characteristics.—There are various parasites which appear to have a causative relation to certain morbid processes. Some of them have been studied in relation to the diseases produced by them; others remain for separate consideration.

VEGETABLE PARASITES.—Parasites infesting the human body belong to the *vegetable* and *animal* kingdoms. The former—or vegetable parasites—are contained in two classes: the Protophyte or Schizophyta; the Zygosporæ. Of the former, the order Schizomycetes contains the most numerous and important of the vegetable parasites, viz., Micrococcus, Bacterium, Bacillus, Vibrio, Spirillum, etc.

To the second order belong the Saccharomycetes: Torulæ and the Sarcina Ventriculi.

MICROCOCCI.—This word signifies *a small berry*, and is applied to a spherical body, highly refractive, of very minute size, never exceeding and often less than $\frac{1}{25000}$ of an inch in diameter. They are seen as isolated dots in active motion, dividing transversely by fission, or form in rows or chains of six, eight, or more, or when united by an intercellular fluid into masses are called *zoöglœa*. They are composed of protoplasm, highly refractile, taking up coloring-matters with facility, but resisting the action of acids and destructive agents generally. They develop most perfectly in fluids containing carbon and nitrogen compounds and a small quantity of free oxygen, but air does not seem very necessary to their growth, since they develop without it under other favorable conditions.

There are various kinds of micrococci. Only those having pathological relations need to be alluded to here. It is quite certain that micrococci are found in connection with various pathological fluids and processes, but it must be admitted, we think, that their exact relation to these conditions can not always be made out. It seems to be well established, however, that if micrococci are entirely excluded, putrefactive decomposition can not occur in animal substances. It was long ago ascertained by Pasteur that every kind of fermentation (putrefactive included) is correlative of the growth and multiplication of some organism. Infective pus owes any virulence it may possess to the presence of these organisms, since it has been shown that fresh pus free from these bodies has no infective property. But there are not wanting facts on the other side, especially the filtration experiments, which seem to prove that the infective property is contained in a substance produced by micrococci in course of their development, and not to their mere presence. The relation of these bodies to some infectious diseases has been shown, according to the best observers. Thus the coccus of pneumonia, now called *pneumococcus* of Friedländer and Fränkel, although not entirely agreed on, must be considered as highly probable; also the coccus of gonorrhœa—*gonococcus*—of diphtheria by Eberth, and a bacillus by Klebs and Löffler, of syphilis by Neisser and Lustgartea, and many other maladies of the character called infective.

Bacteria.—These are rod-shaped, about the $\frac{1}{10000}$ of an inch in length, plain or jointed. They occur in putrescent or fermenting fluids, and vary a good deal in size and shape according to the media in which they are found. In decomposing fluids they are smaller, and in fermenting solutions larger, than the medium size. In the former they are known as *Vibriones*, or in larger and longer rods, as *Leptothrix;* and in the latter they are the well-known *Torulæ.* Some bacteria seem to be entirely without morbific quality, and others, that may not be distinguished from them, contain an intense virus, or, if they do not contain it, have the power to produce it. These pathological bacteria have been discovered in various diseases, as whooping-cough, erysipelas, puerperal fever, and many others. It must be said, however, that only *probability* can be made use of to describe the present state of opinion on the subject.

Bacillus.—The term *bacillus* signifies *a staff*, and is now applied to a filiform bacteria. Bacilli have been found of late in great numbers in certain infectious diseases, notably in splenic fever and malignant pustule. The discovery by Koch of the *bacillus tuberculosis* has taken the first rank as a scientific fact in these times. There is now but little said in opposition to this explanation of the infective property of tubercle, and it has already borne fruit in the treatment of consumption by means to arrest the growth of this parasitic form.

The *bacillus malariæ* of Laveran has been confirmed by late researches, notably those of Professor Osler, of Johns Hopkins University. To this must be added also the *comma bacillus* of Koch, who maintains it as the specific cause of cholera. The discussion on this topic has been warm, not to say acrimonious, but on the whole the position of Koch is supported by an increasing majority of skilled observers.

Spirillum.—This term is applied to the spiral form of bacteria. Cohn, who has made the most careful study of these organisms, and is the highest authority on them, divides these spiral bacteria into three forms: 1. *Vibrio*, which consists of short spiral filaments, very flexible; 2. *Spirillum*, also filaments, but rigid; 3. *Spirochæte*, long, spiral, flexible filaments. The last-mentioned form was described by Obermeier as existing in the blood of relapsing fever during the paroxysms, and his description has been abundantly confirmed. It is sometimes called *Spirochæte plicatilis*, as it is an identical form with that described by Ehrenberg under this name.

Animal Parasites.—Many of the animal parasites have been considered in relation to the diseases caused by them. There are, however, some remaining for examination, which are partly of a practical, partly of a scientific interest. Under the name *chyluria*, mention is made of the *Filaria sanguinis hominis*, a parasite discovered by Dr. T. R. Lewis. An endemic hæmaturia occurs at the Cape of Good Hope and other parts of the African Continent, which is caused by a fluke, called after its discoverer, Dr. Bilharz, of Cairo, the *Bilharzia hæmatobia*. This parasite is found in the veins of the portal system and in the bladder, and causes hæmaturia.

Certain trematode parasites, commonly known as *flukes*, are sometimes found in the human body, notably in the liver. About twenty cases of *distoma hepaticum* have been recorded, several by Professor Joseph Leidy, M. D., of Philadelphia, and they may therefore be encountered in this country. Besides their usual abode in the liver-ducts and gall-bladder, they have been found under the skin behind the ear, under the scalp, and in the sole of the foot.

Of greater practical importance to the physicians of this country, especially of the Southern States, is the *chigoe*, or popularly the *jigger* or *sand-flea*. The popular notion is so far true that this parasite really belongs to the flea tribe, and is named *Pulex penetrans*. It inhabits by preference a dry, sandy soil, and is exceedingly prolific. It deserves to have the adjective *penetrans* appended to *pulex*. They bore into the skin of the feet, especially under the nails and between the toes, where the softer integument permits more ready penetration. The impregnated female, thus deposited under the skin, swells to considerable size, and excites a violent local inflammation, suppuration, and sloughing. The larvæ also, deposited in open wounds and ulcers, excite unhealthy and destructive inflammation. The bones finally become diseased, and toes may slough off, or an extremity may be lost by a necessary amputation.

It follows, from these considerations, that when a jigger penetrates the skin it should be removed with the utmost care, so that no portion of it, or any of its larvæ, be permitted to remain behind. If the parasite is not successfully removed, its destruction by carbolic acid, or sulphurous-acid lotions, should be attempted.

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